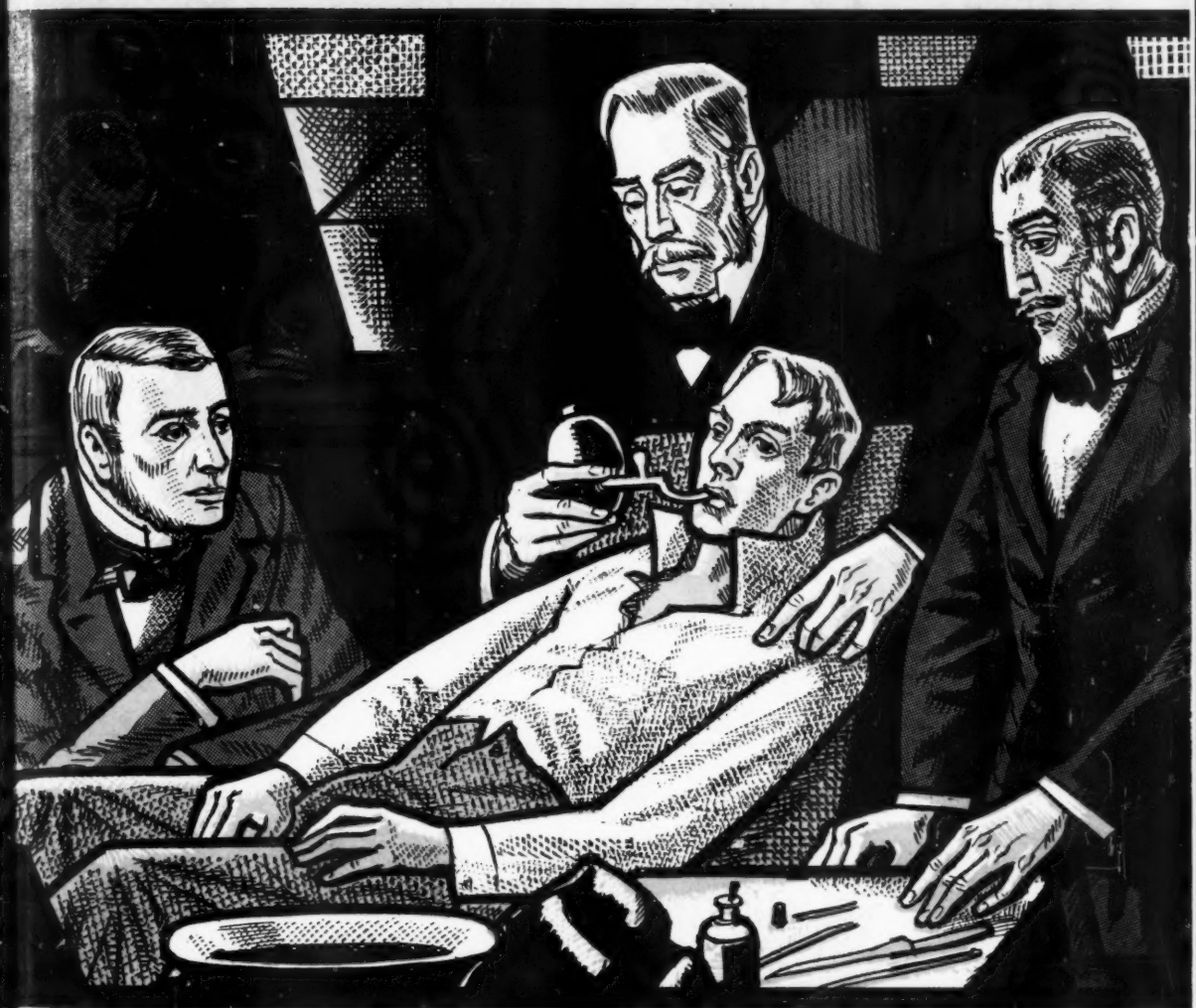
Postgraduate Medicine

RECEIVED
AUG 18 1950
MEDICAL SCHOOL

Vol. 8, No. 1 — JULY, 1950



PUBLIC DEMONSTRATION OF ANESTHESIA

Table of Contents on Pages A-5 and A-

. today's tests, treatments, technics

Nesselrod's Proctology in General Practice

See SAUNDERS Advertisement on Pages 8 & 9

rtisement)

610.5
P872

SPOIL THE APPETITE . . .

CONTROL OF APPETITE is frequently beyond the power of human will, a fact that explains most cases of obesity. Fortunately, appetite can be checked by administration of certain sympathomimetic drugs, such as *Propadrine*® phenylpropanolamine HCl, a development of Sharp & Dohme research notably free of the unpleasant side effects associated with ephedrine. ALTEPOSE tablets, a new formula for control of obesity, provide PROPADRINE HCl, 50 mg. (¾ gr.), to reduce the desire to eat; *thyroid*, 40 mg. (⅔ gr.), to increase metabolism; and DELVINAL® vinbarbital, 25 mg. (⅜ gr.), for mild sedation. ALTEPOSE tablets spare the obese patient the pangs of hunger, making low-calorie diets more acceptable, speed metabolism of excess fat and carbohydrate, and tend to suppress nervous tension and anxiety. The proper dose must be determined for each individual. ALTEPOSE tablets are supplied in bottles of 100 and 1,000. Sharp & Dohme, Philadelphia 1, Pa.

spare the patient

SHARP & DOHME

Altepose® TABLETS

for treatment of obesity

Medical

Postgraduate Medicine

OFFICIAL JOURNAL OF THE INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA — ORGANIZED IN 1915

Editor-in-Chief: CHARLES W. MAYO, M.D.
ROCHESTER, MINN.

Managing Editor: ARTHUR G. SULLIVAN, M.D.
MADISON, WIS.

Contributing Editor: MORRIS FISHBEIN, M.D.
CHICAGO, ILL.

SURGICAL ADVISORY BOARD

Richard B. Cattell, M.D.
BOSTON, MASS.

George W. Crile, Jr., M.D.
CLEVELAND, OHIO

Michael E. DeBakey, M.D.
HOUSTON, TEXAS

I. Ridgeway Trimble, M.D.
BALTIMORE, MD.

Oliver S. Waugh, M.D.
WINNIPEG, MANITOBA

MEDICAL ADVISORY BOARD

Francis G. Blake, M.D.
NEW HAVEN, CONN.

Ray F. Farquharson, M.D.
TORONTO, ONTARIO

William S. McCann, M.D.
ROCHESTER, N. Y.

J. Roscoe Miller, M.D.
CHICAGO, ILL.

James E. Paullin, M.D.
ATLANTA, GA.

Tom D. Spies, M.D.
BIRMINGHAM, ALA.

William D. Stroud, M.D.
PHILADELPHIA, PA.

FOR GREAT BRITAIN

E. R. Cullinan, F.R.C.P.
LONDON

Sir Archibald McIndoe, F.R.C.S.
LONDON

C. Naunton Morgan, F.R.C.S.
LONDON

SPECIALTY ADVISORY BOARD

Brian Blades, M.D., *Thoracic Surgery*
WASHINGTON, D. C.

J. Barrett Brown, M.D., *Plastic Surgery*
ST. LOUIS, MO.

R. Lee Clark, Jr., M.D., *Cancer Research*
HOUSTON, TEXAS

Winchell McK. Craig, M.D., *Neurologic Surgery*
ROCHESTER, MINN.

Franklin G. Ebaugh, M.D., *Psychiatry*
DENVER, COLO.

Walter Freeman, M.D., *Neurology*
WASHINGTON, D. C.

H. Dabney Kerr, M.D., *Radiology*
IOWA CITY, IA.

Julius Lempert, M.D., *Otology*
NEW YORK, N. Y.

Charles F. McCuskey, M.D., *Anesthesiology*
LOS ANGELES, CALIF.

Irvine McQuarrie, M.D., *Pediatrics*
MINNEAPOLIS, MINN.

Hamilton B. G. Robinson, D.D.S., *Dentistry*
COLUMBUS, OHIO

Harry L. Rogers, M.D., *Allergy*
PHILADELPHIA, PA.

Robert D. Schrock, M.D., *Orthopedic Surgery*
OMAHA, NEB.

Bedford Shelmire, M.D., *Dermatology & Syphilology*
DALLAS, TEXAS

Arthur Purdy Stout, M.D., *Pathology*
NEW YORK, N. Y.

Gilbert J. Thomas, M.D., *Urology*
LOS ANGELES, CALIF.

Paul Titus, M.D., *Obstetrics & Gynecology*
PITTSBURGH, PA.

Derrick Vail, M.D., *Ophthalmology*
CHICAGO, ILL.

Postgraduate Medicine is published monthly at Minneapolis, Minn., by IPMA Publishing Co., J. G. Cohen, publisher; G. Victor Lewrie, assistant publisher; William H. Hull, business manager; Fred Couzens, Eastern manager, 420 Lexington Ave., New York 17, N.Y., telephone Murray Hill 4-7752. Advertising representatives: Kenneth Kruse, 859 First National Bank Bldg., Chicago, Ill., telephone Central 6-2711; Duncan A. Scott & Co., Mills Bldg., San Francisco 4, Calif., telephone Garfield 1-7950; Duncan A. Scott & Co., 2978 Wilshire Boulevard, Los Angeles 5, Calif., telephone Dunkirk 8-4151. Publication date: 15th of the month. Subscription rate: $8.00 a year in the United States and Canada; $10.00 in foreign countries. Two volumes a year. Address all editorial and business correspondence to **Postgraduate Medicine**, 516 Essex Building, Minneapolis 3, Minn.

Copyright 1950 by IPMA Publishing Co. Title Reg. U. S. Pat. Off.
Entered as second-class matter February 27, 1947, at the post office at Minneapolis, Minn., under the Act of March 3, 1879. Additional entry at Long Prairie, Minn., under date of April 14, 1948. Printed in U.S.A.

Psychoneurotic Women of Genius

Isadora Duncan, renowned American dancer, was admired throughout the world for her creative ideas and graceful artistry, but estranged her native public through her psychoneurotic eccentricities.

The majority of psychoneurotics have no serious mental illness, but display merely an emotional imbalance which often can be greatly improved by appropriate psychotherapeutic and sedative management. In the treatment of psychoneurosis, particularly agitated, depressed and anxiety states, Mebaral is especially useful when tranquillity with minimal hypnotic action is desired. Sedative dose: Adults, from 32 mg. to 0.1 Gm. (½ to 1½ grains) three or four times daily. Children, from 16 to 32 mg. (¼ to ½ grain) three or four times daily. Supplied in tablets of 32 mg., 0.1 Gm. and 0.2 Gm.

MEBARAL®

BRAND OF MEPHOBARBITAL

TASTELESS SEDATIVE AND ANTIEPILEPTIC • LITTLE OR NO DROWSINESS

Winthrop-Stearns INC.

NEW YORK, N. Y. WINDSOR, ONT.

Mebaral, trademark reg. U. S. & Canada

The Anesthesiologist in the Management of the Surgical Patient

URBAN H. EVERSOLE*

THE LAHEY CLINIC, BOSTON

Progress in anesthesia these last few years has closely paralleled that of surgery, and I believe we as anesthesiologists would not be unduly presumptuous to say that there is an interdependent relationship in that progress.

We all are aware of the fact that the patient benefits when a physician-anesthesiologist is accepted as a consultant on a par with the other consultants. Too often in the past many anesthesiologists have been prone to regard this acceptance as a full consultant as an attitude of mind on the part of the surgeon, and as nothing else. It is more than that. Acceptance as a full consultant implies that the anesthesiologist is not only willing but able to assume that role, that he is prepared to render an opinion that is of value, an opinion that is sought and respected.

Sometimes a vicious circle has developed with reference to the relationship between the surgeon and the anesthesiologist. The anesthesiologist sees himself in a technical position and assumes that he is held in this position by the surgeon, hence he has little incentive to try to develop other fields of usefulness in the care of patients outside of the actual administration of the anesthetic itself. On the other hand, the surgeon sees the anesthesiologist performing a technical job, frequently a very good one, but sees little manifestation of interest on his part to develop fields outside the actual administration of the anesthesia in which he can be of service. Hence, he rightly thinks of him as a technician.

It is easy to be a technician; it allows more time for golf, but it does not provide as much satisfaction as does the development of the many opportunities for service which this specialty affords.

The function of the anesthesiologist during the operation in the maintenance of a satisfactory and safe anesthesia is well known. His ability to provide adequate relaxation for abdominal surgery or to receive the blame when such is not provided is well known by everyone. The other fields of service outside of this are not so familiar. We even find many anesthesiologists themselves who do not appreciate the wide field of service encompassed by their specialty.

We might inquire what some of these fields are in which the anesthesiologist can be of service to surgical patients other than the maintenance of a satisfactory anesthesia. For the sake of convenience I will divide these into the rather obvious classifications of preoperative, operative, and postoperative care.

We might well start with the preanesthetic visit. Too often this has amounted to little more than a visit by the anesthesiologist to order routine preanesthetic medication and perhaps get the patient's address for purposes of billing. It has to be more than that. The anesthesiologist is in a peculiar and very advantageous position from the standpoint of seeing patients preoperatively. He is usually the last person among the consultants who has an opportunity to review the history and the physical findings carefully, and to evaluate the risk of the patient.

*Head of the Department of Anesthesiology, Lahey Clinic, Boston, Massachusetts.

Presented November 1, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

We might say a word about the evaluation of risks. Estimation of the risk of the patient should be made independent of the operation to be performed. Although the proposed surgical procedure has nothing to do with the risk, it may have a great deal to do with the decision as to whether the operation is done at all.

The anesthesiologist then should interview the patient. This is often glossed over or completely neglected. This interview is very important from the patient's standpoint, in obtaining his confidence, in assuring him that he can be carried safely through the procedure, and that he need not anticipate too much discomfort after operation. An interview can be of great value in clearing up many misunderstandings. How many of us have heard the nurse on the floor say, "This patient will not take spinal anesthesia," or "This patient will not take ether"? A talk with the patient, explaining to him the reasons for the choice of this or that anesthetic is usually all that is necessary. It is rare for the anesthesiologist or the surgeon to have to change his plans because of any wishes on the part of the patient. The patient who says he will not have spinal anesthesia usually means he merely wants to be unconscious.

The anesthesiologist is in a very advantageous position for evaluating preanesthetic medication. In fact, he is the only one on the surgical team who can properly evaluate because he is the only one who at first hand sees the effect of the medication he has ordered.

The prime responsibility for the selection of the anesthetic should rest with the anesthesiologist. This does not mean he should always make the selection without consultation with the surgeon; in fact, it is an unwise anesthesiologist who does not consult with the surgeon, particularly for an unusual procedure.

We may now turn our attention to some of the fields not so directly related to the operation itself in which the anesthesiologist can be of service.

He is used to blocking nerves with anesthetic agents for the production of anesthesia. This experience can be put to good use in diagnostic and therapeutic nerve blocks for the evaluation of many types of obscure pains, the treatment of peripheral vascular disease, and so forth. Frequently the blocking of certain nerves is of value as a prognostic sign as to what can be expected from surgery. As examples we might mention lumbar sympathetic block in peripheral vascular disease and stellate ganglion block for upper arm and neck pain.

THE following review of the history of one patient illustrates the importance of a series of diagnostic nerve blocks prior to surgery: A 26-year-old nurse complained of pain in the left side in the region of the kidney which had been sufficient to prevent her working for several weeks. This pain was thought to be sympathetic in origin. A series of sympathetic nerve blocks was performed, beginning low in the lumbar region. By proceeding slowly, one segment at a time, we were able to determine that blocking of the tenth and eleventh thoracic segments completely relieved the pain and blocking of any other segment did not. Surgical removal of these two segments of the sympathetic chain resulted in complete relief.

I cite one instance of stellate ganglion block which was of definite therapeutic value. A 65-year-old man who had had a cerebrovascular accident was seen about six hours later by the anesthesiologist, at which time he had some recovery of the paralysis of his left side but had an incapacitating headache. Stellate ganglion block was done on the right side with complete relief of his headache, which did not recur through the rest of his stay in the hospital, during which time he obtained almost complete return of function of his left side.

A series of suprascapular nerve blocks at the suprascapular notch may be valuable in the treatment of obscure pains in the shoulder.

We are all familiar with the severe pain of acute pancreatitis. This can be relieved by epidural block at the level of the seventh, eighth, and ninth dorsal segments. If a fine ureteral catheter is placed into the epidural space near this level continuous relief can be obtained for an indefinite length of time.

The anesthesiologist can well assume the full responsibility for the general condition of the patient during operation, and I believe that in most instances the welfare of the patient is better served if he does carry this responsibility. This means that he must be responsible for all supportive therapy such as stimulants, fluids, and blood. This presupposes that he is aware of all the problems associated with the collecting and storing of blood and blood derivatives. If he is responsible for the administration of blood his is then the final check to be sure that the

wrong type of blood is not given. Furthermore, the anesthesiologist should be familiar with accepted methods of treatment in the event this unfortunate accident occurs.

In most instances a clerical error somewhere along the line is responsible for the inadvertent administration of mismatched blood. One important safeguard against this error is a double check accomplished by drawing separate samples of blood from the recipient on different occasions for the typing and cross-matching. One technician should draw the sample for typing and another technician at another time draw the sample for cross-matching. We had this unfortunate accident occur in a vigorous 19-year-old male upon whom a rather shocking orthopedic procedure was being performed. He had received 500 cc. of incompatible blood and it was not discovered that it was the wrong blood until a second transfusion was ordered. According to our custom, this second blood was cross-matched with the first and at that time the discovery was made. Fortunately, this patient survived, partly because of the vigorous treatment he received, but in large part owing to his young and vigorous physique.

One word about treatment following an incompatible blood transfusion. If the patient is under anesthesia, the early symptoms of backache, chills, rise in temperature, tingling sensations, constrictions in the chest, and restlessness are not apparent.

Treatment of hemolytic transfusion reactions is directed along two major lines: (1) urinary alkalinization to prevent the blocking of the uriniferous tubules by precipitated acid hematin, and (2) correction of the secondary anemia and shock which may accompany this condition.

The urine is kept alkaline (*p*H 7.5 to 8) by the oral administration of sodium bicarbonate. If oral medication is not possible sodium racemic lactate or sodium bicarbonate should be given intravenously. The urine must be kept alkaline until there is no evidence of hemoglobinuria. With this high degree of alkalinity the patient should be watched carefully for signs of alkalosis. The early administration of 30 to 50 cc. of A and B specific substances may be helpful in neutralizing the agglutinins that are in the recipient's serum. This has a tendency to slow down the agglutination of the remaining incompatible donor cells. General supportive treatment should be carried out. This may require the administration of compatible whole blood to combat the hemolytic anemia that is so often associated with transfusion reactions.

The anesthesiologist is familiar with direct laryngoscopy and bronchoscopy and can be of great help at times in the operating room by passing a bronchoscope, particularly when patients have aspirated vomitus. We have seen one patient in whom complete respiratory obstruction developed during the course of an operation owing to expression of undigested food from an esophageal diverticulum which was being removed surgically. This completely blocked off both main stem bronchi and necessitated bronchoscopy during the operation.

The anesthesiologist should be able to anticipate emergencies. No longer should we see frantic efforts being made in an attempt to insert a needle into a collapsed venous system of a patient in shock. This is already taken care of; needles are in place and the anesthesiologist is prepared to take care of any type of circulatory emergency which may arise.

Perhaps the most dramatic of the emergencies which may arise during the course of operation is cardiac arrest. Cardiac arrest is, fortunately, an infrequent complication, but it is a very dramatic one. It must be distinguished from a carotid sinus reflex. We believe that every form of asystole which occurs during operation should be considered as a cardiac arrest, and steps should be taken immediately to combat it. Even with a well set-up system it is impossible actually to institute treatment under thirty seconds, and if by the time the thirty seconds have elapsed the heart has not started, it may then be considered to be a cardiac arrest. We feel that every operating room should be equipped with syringes, stimulating drugs, procaine, heart needle, and so forth, and they should all be sterile and together in one place, ready for instant use. Such a kit is present in all of our operating rooms. Immediately after the diagnosis is made, 4.75 cc. of procaine with 0.25 cc. of epinephrine is drawn into the syringe. This is immediately injected into the cavity of the heart after aspirating to be sure the needle is within the cavity. The needle is inserted through the fourth or fifth intercostal spaces on the left.

During this time the surgeon is preparing to massage the heart. If he is operating in the abdomen, he opens the diaphragm and inserts his hand into the chest cavity. If the operation is

elsewhere than in the abdomen, or in the chest, the surgeon has a choice of routes, and obviously, through the chest is the better route. This can be done very easily without chest instruments, merely by making an incision through the fourth or fifth interspace, and sectioning the costal cartilage near the sternum below the intercostal space opening. This allows adequate room for massage.

We have had 15 such cases in the last twelve years. We have been able to start all of the hearts, but only 8 of the patients survived as normal individuals. We have at our disposal a maximum of five minutes, and probably only three or four minutes to establish circulation if we expect to have a normal patient.

I would like to cite one case as an example of cooperative effort in the care of a serious risk patient. A 64-year-old man was admitted to the hospital for treatment of severe bronchitis superimposed upon an emphysema which had been serious enough to necessitate his stopping work many years before. About five days after he entered the hospital a severe gastric hemorrhage suddenly occurred from what was thought to be a quiescent gastric ulcer. His hemoglobin dropped abruptly from 13.8 to 6 gm. per 100 cc.

During the next four days he was given a total of 11 pints of whole blood. The highest figure reached by his hemoglobin during these four days was 7 gm. per 100 cc. It became obvious that surgery must be resorted to if his downhill course was to be reversed. At operation the hemoglobin was 5.2 gm. with a red blood cell count of 1,500,000.

The operation (gastric resection) was performed under cyclopropane-curare anesthesia. After insertion of the endotracheal tube, a large amount of purulent secretions was aspirated from the air passages. A total of four transfusions was given during the operation, at the end of which his blood pressure was 130 mm. systolic and his pulse rate was 120 per minute. Because of the purulent secretions in the tracheobronchial tree he was submitted to aspiration bronchoscopy at the end of the operation. This patient required no more blood during an almost uneventful convalescence. He was discharged from the hospital twelve days after his operation with a hemoglobin of 13.2 gm. per 100 cc. and with his pulmonary condition greatly improved.

The anesthesiologist watches the patient through his surgical course and knows his reaction to supportive treatment, hence he is the natural consultant for supportive treatment during the early postoperative period.

We are all familiar with the tremendous part played by atelectasis in postoperative pulmonary complications. Our old diagnosis of ether pneumonia was really atelectasis due to blocking of a bronchus or bronchioles by secretions, with absorption of the air distal to the plug, collapse of that area, and subsequent infection. The anesthesiologist with his experience in laryngoscopy and bronchoscopy can be of great value in the treatment of these patients by removing the offending secretions by aspiration. Once a patient has demonstrated his inability or unwillingness to remove secretions by coughing, an attempt should be made to remove them by aspiration. Many times this can be done by passing a number 20 or 22 French urethral catheter into the trachea and applying suction. If this is not adequate the patient should be submitted to bronchoscopy.

I am sure that many of you, if you were to make a survey of oxygen therapy as it is carried out in your hospital, might get the surprise that we got when we surveyed our routine for inhalation therapy. In some instances patients were being submitted to the discomfort and expense of an oxygen tent without actually increasing the oxygen content of their atmosphere any appreciable amount. The assumption of responsibility for this type of therapy by someone who is interested in gases will almost always result in improvement of that service.

FINALLY, I might say that we think there is a definite place for the specialty of anesthesiology. The anesthesiologist has something to contribute to the care of the surgical patient over and above his ability to conduct the patient safely through his anesthetic course and provide satisfactory working conditions for the surgeon. In order for him to do this he must be on a full consultant basis. To be on a full consultant basis he must be capable of assuming that role, and he must be accorded that role by the surgeon once he has proved his ability to assume it.

I think Gillespie has very well summarized the ideal relationship between surgeon and anesthesiologist. He says: "The essentials of their relationship are mutual professional confidence, understanding, frankness, honesty and courtesy, with fair-mindedness on both sides."

Alcoholism and the General Practitioner

JOSEPH HIRSH*

COMMITTEE ON PROBLEMS OF ALCOHOL, NATIONAL RESEARCH COUNCIL, WASHINGTON, D. C.

THE seeming recent preoccupation on the part of the general practitioner with the "whole organism" is misleading. Truth of the matter is that he, in his unique position as a sort of company commander, has always had to deal at first hand "in the field" and often alone, with the "whole organism." Occasionally, in the matter of strategy he will refer some particular problem to higher headquarters—to the G-2 of pathology or some other staff specialist—but in the matter of tactics he remains, first and finally, the key man in dealing with the "whole organism" and with the factors which influence it in health and disease.

General medicine—and the original subspecialties which grew out of it, hygiene and pathology—pioneered the importance of living and working conditions and of emotional factors in the predisposition to, the process, the treatment, and control of disease. In Bernadino Ramazzini *(De Morbis Artificium Diatriba**)* and Johann Peter Frank *(System Einer Vollstandigen Medicinischen Polizey)* we clearly find evidence of such general medical leadership. In our own time Osler typified this orientation in innumerable ways and instances, the classic of which is his reference to tuberculosis as a social problem with medical manifestations.

In a recent article on the integration of psychiatry in undergraduate medical education, Malamud stated, "The physician in general practice is the one who is most likely to see the earliest manifestations of what may eventually develop into the most serious psychiatric disturbances, and he will have the best opportunity of preventing them if he is properly prepared (oriented and sympathetic) for the recognition of the early danger signals."[1]

The rationale for this presentation, therefore, is based upon two premises: First, alcoholism is a medical problem—usually of the total personality and not any single segment—and the alcoholic is a sick person. Second, because of the traditional leadership of the general practitioner in the area of total personality and its needs as well as his position in the vanguard against disease, his continuous and sympathetic attention is needed to provide effective care for the many tens of thousands of patients now abandoned to the ministrations of courts, jails and public shelters, and to prevent the serious deterioration—physical, mental, and social—common to all of them.

In stating this latter premise the author is not unmindful that the alcoholic, even in his casual relationships with medicine, has often earned for himself the unenviable reputation of being a contumacious nuisance and an unredeemably hopeless patient. Nevertheless his problems and needs are so essentially medical that he will always remain a challenge to the profession. He can not be ignored nor foisted upon any other group. Happily, in recent years medicine has begun to face his problems squarely, with the result that many thousands of patients once abandoned as hopeless are now useful and productive citizens of society.

*Executive Secretary, Committee on Problems of Alcohol, Division of Medical Sciences, National Research Council, Washington, D. C.

**An excellent annotated English translation of this text by Wilmer Cave Wright was published by the University of Chicago Press in 1940.

SIZE AND NATURE OF THE PROBLEM

Statistically, alcoholism is a relatively rare syndrome among social drinkers. In the United States there are estimated to be in excess of 60,000,000 social drinkers. Of these, approximately 3,000,000 consume alcoholic beverages excessively.[2]* Although this group generates the largest proportion of problem drinkers, they are still in the elective stage of their drinking patterns. The effective rehabilitation of these "patients" by the Yale Plan Clinics and, in recent years, by other clinics and by Alcoholics Anonymous will be a source of encouragement to those practitioners interested in preventive medicine in the broadest sense, who will find many of these potential alcoholics amenable to treatment.

There are finally the problem drinkers themselves—three-quarters of a million of them—who are sick to begin with, become sick in their dependence upon alcohol, or end up with physical and mental damage.[4]

Considering the total exposure rate to alcoholic beverages, statistically, alcoholism is a relatively minor problem. Considered in comparison with the aggregate problem represented by tuberculosis, cancer, and infantile paralysis, today alcoholism looms as one of the country's major problems in the United States.

ETIOLOGY OF ALCOHOLISM

Though the etiology of alcoholism is not known, in recent years we have happily begun to move away from the traditional view that the chemical, C_2H_5OH, or a "weak will" is the responsible factor. Until recently many psychiatrists have taken the equally narrow view that unremitting, excessive drinking is attributable largely to a neurotic pattern of behavior.

There is a growing body of evidence to suggest that an abnormal psycho-physiologic reaction to alcohol on the part of some individuals, not shared by most social drinkers, may be at the bottom of the disease-complex of problem drinking. The changes both in personality and in the biochemistry of the body metabolism of the problem drinker suggest that there is considerable validity to this premise.

That there may be an inherent constitutional predisposition is clearly evident in many cases.[5] Lemere and his associates[5] have found excessive drinking in the family background of the "alcoholic addict" to be four times as frequent as in the case of normal drinkers.

*A number of investigators, notably Jellinek[2] at Yale and Riley and Marden[3] at Rutgers, have published significant data on male-female ratios and the influence of urban, rurban and rural conditions of living, educational, religious, marital, and other factors in normal, excessive, and problem drinking.

THE ALCOHOLIC IS A SICK PERSON

The nosology of alcoholism is still imperfect and arbitrary. Among those who have pioneered in the problem of classification and definition Bowman and Jellinek[6] stand out. A number of clinicians—both psychiatrists and internists—have also tackled this problem. From the welter of groups and subgroups they have devised three main alcoholic types: the symptomatic, the true addict, and the secondary addict.

Estimates vary, but it is generally conceded that symptomatic alcoholics comprise between 40 and 60 per cent of all inebriates. By definition the symptomatic alcoholic is a person whose pathologically excessive and frequently (self) destructive drinking is a symptom of some underlying mental or physical illness, inadequacy, or defect. Symptomatic alcoholism is often a concomitant or expression of epilepsy, psychosis, psychoneurosis, mental deficiency, or psychopathic personality.

By definition, therefore, the symptomatic alcoholic is a sick person in need of medical care with and without reference to his consumption of the chemical, alcohol. Treatment of these patients, therefore, solely in terms of depriving him of alcohol, is of doubtful value unless concomitantly his basic illness is treated.

Relatively little is known of the true addict, of the etiology and process of his addiction. In his psychodynamics of alcoholism, Knight[7] views the true addict as an "essential alcoholic." Under the leadership of Williams,[8] a number of investigators currently working in this field view this type of alcoholism as an unusual metabolic reaction, one of the "inborn errors of metabolism." Susceptibility to alcoholism, according to this theory,[8] therefore, is endogenously determined by the individual's "metabolic personality" at birth, or as it is modified during important growth periods.

As is often pointed out, in spite of potential and frequently inevitable constitutional damage, and despite personality deterioration with dire social consequences, alcohol is for the true addict an inevitable, a necessary, yet unmanageable poison.

Whether or not one is willing to accept defects in the "biochemical personality" predisposing to alcoholism as *a disease,* the end results of excessive drinking and the coextensive products of poor living (true for all three types of alcoholics), such as the avitaminoses, Korsakoff's syndrome, Wernicke's syndrome, the so-called alcoholic hallucinoses, delirium tremens, are clearly pathologic conditions and are the concern of the medical profession.

Finally, the secondary addict *appears* to be a reasonably well-adjusted person psychologically, to begin with. This is clearly not the case in the symptomatic addict either from the psychologic or from the physical point of view. Knight[7] describes the secondary addict as a reactive alcoholic, in whom

we find strong psychosocial and situational factors predisposing to excessive drinking—the job that is inadequate or too challenging, the marriage that is too much, the traumatic illness, accident, or loss of a loved one. The drinking patterns in these individuals are irregular and sporadic and, as their name suggests, are almost always a reaction to precipitating situational factors or reality stress.

During the period which has been defined by some as habituation in a psychologic sense, and by others with a physiologic point of view, as a biochemical alcoholic orientation, distinct personality changes—and possibly physical changes—take place in the secondary addict. Whatever the etiology of secondary addiction—whether it be psychosocial to begin with and biochemical incidentally—the end-products in secondary addiction problem drinking are often physical *and* mental deterioration.

THE ADDICTIVE PROCESS

Someone has made the observation that the unique characteristic of alcoholism as a disease is due to the fact that no *single* pathologic organism or agent has been isolated as the etiologic factor. This same anonymous clinician has wisely stated that there are as many diseases of alcoholism as there are alcoholics. In other words, alcoholism is more a protean constellation than a single disease. Its etiology, its characteristics and form, its process vary widely from individual to individual. Clearly, in a disease context, it can not be compared with tuberculosis or diphtheria, whose etiologic agents are known, whose pathologic patterns are more or less consistently uniform, whose end results are known or can be estimated.

Despite these limitations, it is possible to describe in a generic sense the addictive process. As herein used, the term "addiction" refers not merely to the psychologic process but also to the physical process of conditioning.

In general, it may be said that the symptomatic alcoholic and true addict make a poor adjustment to their total environment prior to their addiction while the secondary addict appears to make a reasonably good adjustment at least on an interval basis. In the first instance we find that there is no problem in tracing the character of the addictive process since it is as regular and as inevitable as the tides.

Not so with the secondary addict who, as a rule, starts out as a social drinker and, due largely to exogenous predisposing, precipitating, or situational factors, becomes an excessive though normal drinker, i.e., in the sense that he is still in the elective stage of whether or not he will continue to drink excessively.

The causative factors may build up to such an extent, however, that the first "lost week end" eventuates, to be followed by guilt and self-recrimination. This is the real starting point of the unremitting phase and more "lost week ends" ensue.

Frequently at this point the general practitioner may be consulted. His attitude towards the patient, who is generally on the defensive psychologically, is of paramount importance. The patient, having experienced one or several alcoholic sprees, cannot be considered to be a true addict; he is still in the prodromal stage. Medicine can salvage him before he gains speed along this erratic road.

The experience of the Yale Plan Clinics and Alcoholics Anonymous in recent years has shown that younger groups of alcoholics can be successfully exposed to "preventive therapy."[10] This is as should be expected. The younger the patient the less likely is he to have deep personality involvements, the less fixed are his drinking and other social patterns. While success in treatment and permanency in rehabilitation are generally in inverse proportion to the degree of personality involvement, one of the real jobs of medicine is to "educate" the younger patient to the desirability of medical care. Too many of them who have neither suffered enough as a result of their personality defect or their excessive drinking are difficult to persuade to abstinence or therapy. They still carry the dream of the elective character of their drinking habits and general patterns of living.

ATTITUDE OF THE PATIENT

The receptivity of the patient to medical care is essential for successful treatment. The prognosis for treatment initiated not for himself but to satisfy someone else is poor. Unless the patient really wants to be helped and makes an effort at the initiation of treatment to stop drinking, it has been found that treatment is useless. His desire to recover, to be rehabilitated, is all-important.

Another important element in the psychology of the patient—one which can be used as a constant and powerful instrument in therapy—is the fact that, in coming to the physician for care, something (possibly for the first time in his experience) is really going to be done to him and for him. The very fact that the physician is sufficiently interested to consider him a patient instead of an object of moral turpitude is in itself an important psychologic factor in therapy and points up the need for a sympathetic approach to him.

THE GENERAL PRACTITIONER AND TREATMENT

Should the biochemical hypothesis on alcoholism prove to be correct, it is entirely possible that alcoholics, or some of them at any rate, may, in the future, yield to chemical treatment. Present-day

treatment of alcoholism, however, is primarily psychologic, with heavy sociologic and educational undertones.

For many alcoholics the whole process of living is a difficult and complicated one; it is frequently the reason why they become alcoholics in the first place. This fact must be constantly borne in mind during therapy and calls for the very best in medicine in the way of patience, compassion, and understanding. It calls for the same sufferance and understanding towards recidivism as to recurrences of other psychologic or somatic ailments. It demands that the goals set for the patient in therapy—whether they be abstinence, adequate diet, new interpersonal and group relations, new recreational activities, or vocational pursuits—be spaced one at a time, rarely two or more in combination. If the problems in therapy become too great for the patient, he will more often than not abandon it.

Within this framework the general practitioner faces a number of therapeutic problems and opportunities. The immediate challenges of acute intoxication and its medical management have been discussed elsewhere.[11] The existence of subacute, acute, and chronic conditions[9,12,13] common to many alcoholics must be corrected not only as intrinsic problems but also because these patients rationalize the presence of pain, fatigue, debility, and insomnia to justify their recidivism. The correction of these somatic problems—the gastrointestinal disturbances, the malnutrition and anemia, the vitamin deficiency diseases—is not only a prerequisite to dealing with the personality problems of the patient but in itself makes a surprising difference in the character and content of these problems.

In his strategic position in the vanguard of medicine, the general practitioner can assess the need for all types of specialist care. Alcoholics in need of psychiatric referral present a number of very real problems and challenges, not the least of which are the seriousness of personality deviants in the alcoholic and the difficulty in making a diagnosis. Indications in deciding upon psychiatric referral for alcoholic patients are previous "nervous breakdown," history of typical psychopathy, well defined mental illness—anxiety state, hysterical reaction, delusions, hallucinations—epileptic features, and abnormal electroencephalogram, to mention a few.

A second set of problems facing the general practitioner in making psychiatric referrals for alcoholic and other patients in need of such specialist care is its general unavailability both in terms of trained psychiatric personnel and facilities. Where possible, referrals should be made to a teaching hospital or to well-staffed, well-equipped institutions in which such long-term *in-service* care and study as may be indicated, can be effected. Though this persists as one of the key blocks to medical care, more general and special hospitals than ever before are beginning to open their doors to these patients.

Government likewise, in recent years, has begun to take a more active role in recognizing alcoholism as a disease and in providing medical and institutional care for patients so afflicted. At the close of World War II only 2 states had established such programs. By 1947 8 states were engaged in studying the problems, in providing medical care for alcoholics,[19] or both. By July, 1949, 22 states had established such programs. While many of them are still too new either to meet the heavy demand or to provide immediate relief to the general practitioner in the matter of referrals, there is another agency to which he can turn—Alcoholics Anonymous.

Alcoholics Anonymous, a society of alcoholics who banded together in 1938 to help themselves, now has over 1,500 local chapters or groups throughout the United States and abroad and a membership of over 80,000 men and women. An index of its effectiveness can be found in the fact that its members—once the economic and social debits on society's ledger—are now contributing over $150,000,000 worth of goods and services to society. Once the country's notable emotional and physical dependents, its members are now self-sufficient and self-sustaining.

Operating on the principle that only those alcoholics can be helped who want to be helped, Alcoholics Anonymous welcomes all comers. It has no membership requirements beyond this self-help principle. Its "Twelve Step" program of self-analysis, help, and rehabilitation attracted, in the early days, only diehard, confirmed alcoholics; today it attracts many pre-alcoholics. The general practitioner will find Alcoholics Anonymous helpful in many cases. Addresses of local groups can be found in many communities' telephone directories or through the national office of the Alcoholic Foundation, 420 Lexington Avenue, New York.

If deep psychiatric care does not seem indicated, the general practitioner is uniquely qualified to uncover the more obvious and chronic psychosocial problems of the patient. In the traditional doctor-patient relationship, in creating an aura of assurance and establishing patient rapport, he is singularly situated to engage in psychotherapy.

Psychotherapy in the treatment of alcoholism in general practice, primarily of the secondary addict, is essentially a problem in human relations. As in any other problem, definition, delimitation, and evaluation, likewise critical elements in diagnosis, are *sine qua non* to solution. An accurate estimate of the patient can no more be made in times of acute stress than in acute intoxication. The character and degree of psychosocial maladjustment and

neurotic involvement, therefore, can be assessed only during periods of abstinence, when the patient is in a "dry state."

For sound somatic reasons and to permit early personality assessment and the institution of psychotherapy, abstinence is among the first orders of "business." There are few, if any, contraindications to immediate and complete withdrawal.

Once "dry" and symptomatic treatment has been started, ample opportunity exists to size up the patient. If he appears to be reasonably stable and responsible and has successfully made essential social adjustments aside from his drinking pattern, deep analytic investigation and treatment may not be indicated. The truth of the matter is that many clinicians have found that most secondary addicts do not show major evidence of neuroses when they are not drinking.

As part of the personality assessment, evaluation of psychosocial factors predisposing to or precipitating excessive drinking is essential. Therapy is directed at the maintenance of abstinence, for only one drink for the alcoholic is traditionally the trigger mechanism blasting the way for another spree. In the process of achieving and maintaining a goal of sobriety, the patient must be taught to understand that one drink for him is *the* one too many.

Equally vital in the therapeutic process is the joint examination by the physician and patient of the patient's personal capacities—intellectual, emotional, physical, and social—as well as his interpersonal relationships in the home, on the job, in the community. The patient must be taught to recognize, understand, and act within his capacities and limitations, not to resist them or fight them, not to reject them or attempt to escape from them via the neck of the bottle. Therapy at this level involves three R's—reassurance, reeducation, and often redirection.

The practitioner can profitably engage in substitutive treatment aimed at developing new pursuits and activities for former ones. While such treatment rarely touches underlying personality conflicts, it does afford opportunities for new and successful orientations on the part of the patient in environments less hostile and inimical than his previous ones. These opportunities are as much a part of the therapeutic process as an ℞ and are often successful antidotes for C_2H_5OH.

Substitutive treatment is more than the creation of new interests and activities *per se*. It is directed at the development of new and meaningful emotional experiences from these new interests. If alcohol has meaningful content, either positive or negative, for the alcoholic, withdrawal leaves a void in his life. Here then lies a therapeutic opportunity—substituting religious, AA, hobby, or other satisfying emotional and intellectual experiences. Discovering the precise and applicable emotional appeals—which vary from patient to patient—is one of the fundamental challenges facing the practitioner.

Finally, a word about drug therapy—including aversion or conditioned reflex treatment. The literature, ranging from the reports of Lemere and Voegtlin[5,14] and his associates of ten years ago up to and including those on *antabuse*, lends encouragement to those eager for a quick, specific remedy.

Unfortunately, drug therapy is based upon the false premise that the core of the alcoholic's problem is alcohol. Condition him against the substance and *ergo* he is cured. Experience has shown, however, that while he may break with the substance—and at that for no guaranteed period—he is almost always left with those personality problems which underlay his excessive drinking in the first place and most assuredly will insure the recapitulation of the cycle. In recent years, therefore, psychotherapy has been consistently urged as a concomitant to insure effective, lasting conditioning with drugs.

In summation, then, we can state the role of the practitioner in relationship to the alcoholic as follows: When a patient finally decides to stop drinking excessively, he often cannot do so without outside help. The role and responsibility of the general practitioner is clearly one of giving this help, for experience has shown that more often than not the patient is worth helping.

REFERENCES

1. Malamud, W.: Psychiatry in undergraduate medical education. J.A.M.A. 140:1-5 (May) 1949.
2. Jellinek, E. M.: Recent trends in alcoholism and in alcohol consumption. Quart. J. Stud. Alc. 8:1-42 (June) 1947.
3. Riley, J. W., and Marden, C. F.: The social pattern of alcoholic drinking. Quart. J. Stud. Alc. 8:265-273 (September) 1947.
4. Carlson, A. J., and Hirsh, J.: Chronic alcoholism—a public health problem. Health News 26:3-5; 14-15 (January) 1949.
5. Lemere, F., Voegtlin, W. L., Broz, W. R., O'Hollaren, P., and Tupper, W. E.: The conditioned reflex treatment of chronic alcoholism: VIII. A review of six years' experience with this treatment of 1,526 patients. J.A.M.A. 120:269-271 (September) 1942.
6. Bowman, K. M., and Jellinek, E. M.: Alcohol addiction and its treatment. In Jellinek, E. M. (Ed.): Effects of Alcohol on the Individual: Alcohol Addiction and Chronic Alcoholism. New Haven, Yale University Press, 1942, pp. 3-80.
7. Knight, R. P.: The psychodynamics of chronic alcoholism. J. Nerv. & Ment. Dis. 86:538-548 (November) 1937.
8. Williams, R. L.: The etiology of alcoholism: A working hypothesis involving the interplay of hereditary and environmental factors. Quart. J. Stud. Alc. 7:567-587 (March) 1947.
9. Jelliffe, Norman: Vitamin deficiencies in chronic alcoholism. In Jellinek, E. M. (Ed.): Effects of Alcohol on the Individual: Alcohol Addiction and Chronic Alcoholism. New Haven, Yale University Press, 1942, pp. 173-262.
10. Hirsh, J.: The Problem Drinker. New York, Duell, Sloan & Pearce, 1949, page 211.
11. Sharp, L. I.: The treatment of acute alcoholism by the general practitioner. Westchester M. Bull. 14:9-11 (July) 1946.
12. Smith, J. J.: A medical approach to problem drinking; preliminary report. Quart. J. Stud. Alc. 10:251-257 (September) 1949.
13. Voegtlin, W. L.: Limitations and adjunctive therapies in treatment of chronic alcoholism. M. World 65:165-168 (April) 1947.
14. Lemere, F., Voegtlin, W. L., Broz, W. R., O'Hollaren, P., and Tupper, W. E.: Conditioned reflex treatment of chronic alcoholism: VII. Technic. Dis. Nerv. System 3:273-247 (August) 1942.

Acute Appendicitis Complicating Pregnancy

CHARLES I. WARFIELD*

GEORGETOWN UNIVERSITY MEDICAL SCHOOL, WASHINGTON, D. C.

ACUTE appendicitis complicating pregnancy presents to both the surgeon and the obstetrician a serious situation endangering the lives of both the fetus and mother. Recent contributions in the fields of therapy (antibiotics, intravenous fluids and blood in large amounts, early ambulation, improved surgical and obstetric technics, etc.) seem to warrant still further evaluation of this problem if we are to lower the fetal and maternal mortality and subsequently achieve the ultimate in treatment.

This report is undertaken to review the material presented in the literature for the past thirteen years and to present 5 additional cases coming under the direct observation of the author within one year.

HISTORICAL

Acute appendicitis was established as a definite entity by Fitz in 1886. The first case report of acute appendicitis as a complication of pregnancy was made in 1894 by Munde. Since that time a great deal of literature has been presented in regard to this complication of pregnancy; however, relatively few authors have presented large series or numerous personal cases.

PATHOGENESIS

The pathogenesis of the disease depends upon the fact that the appendix is a relatively long, blind diverticulum with a narrow lumen which is easily occluded. Interference with the blood supply is also an important factor, since the appendicular artery is a terminal vessel without anastomosis and in pregnancy the physiologically increased vascularity in the pelvis and lower abdomen may further hinder the blood supply to the appendix, thus predisposing it to the development of phlebitis and thrombosis. It is generally thought that the lessened resistance of the pregnant woman to infectious and toxic processes may be a contributing factor.

INCIDENCE

This has varied greatly with different observers, but generally all agree with Crossen that approximately 80 per cent of all cases of acute appendicitis complicating pregnancy occur during the last six months of gestation.

Norton and Connell reported 1 case of term labor complicated by acute appendicitis in 20,000 deliveries at the Margaret Hague Maternity Hospital, while Mussey and Crane reported 2 per cent of pregnancies complicated by appendicitis, and Schmid 1 per cent of pregnancies with this complication. S. A. Cosgrove has reported 1 case of acute appendicitis in 1,380 deliveries at the Margaret Hague Maternity Hospital. Findley has stated that 70 per cent of all women who have appendicitis without operation will suffer from the disease during subsequent pregnancies. The 5 cases which came under my direct observation and which are presented in this report occurred in 1,000 deliveries.

*Department of Obstetrics and Gynecology, Georgetown University Medical School, Washington, D. C.

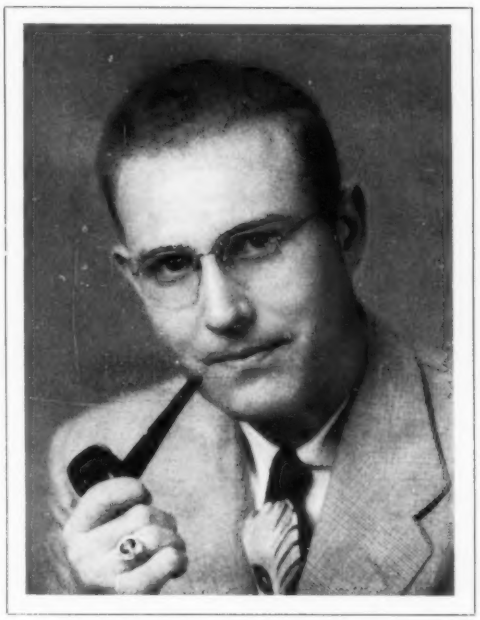
Statistical studies of the larger reported series showed that the gangrenous and ruptured types of appendicitis were respectively 5.5 and 1.5 to 3.5 times as frequent in the pregnant as in the nonpregnant state.

SIGNS AND SYMPTOMS

All the accepted classical clinical signs and symptoms are present with the extremes being the finding of only one or two signs and symptoms to encountering the entire gamut in a typical textbook picture. These signs and symptoms, however, as well as the associated laboratory findings, are considerably altered and even may be masked in pregnancy. Fueth in 1905 was the first to call attention to the anatomic displacement of the cecum and the appendix during pregnancy. Usually, lateral and cephalic displacement begins with the end of the twelfth week and by the twenty-sixth week, the appendix and cecum become abdominal organs. Baer, Reis, and Arens in the United States, and Guthman and Staehler in Germany, substantiated these findings by x-ray studies. This being so, the pain, tenderness, rebound tenderness, and muscular spasticity will progress upward on the abdomen, and laterally towards the right flank as gestation progresses from the twelfth through the twenty-sixth week.

Virchow's "physiological leucocytosis" of pregnancy has been reported by various authors to be between 8,700 and 30,000. In Kreig's series of 51 cases of acute appendicitis in pregnancy the white blood counts ranged from 8,000 to 31,000. In one-third of the cases, the white blood count was below 14,000. The polymorphonuclear count ranged from 68 to 95 per cent with two-thirds of the cases being 80 per cent or less. In the author's 5 cases the white blood cell count ranged from 8,500 to 28,000 and the polymorphonuclear count range very closely approximated that of Kreig.

Vogt reported a progressive increase in the sedimentation rate during pregnancy ranging between 23 and 32 mm. per hour in the ninth month. It returned to the upper limits of normal approximately six weeks postpartum. It is the observation of the author that in appendicitis complicating pregnancy there has been no proportionately significant increase in the "physiological increase" reported by Vogt, and thus, while the procedure is carried out, it very infrequently aids in establishing a diagnosis.



CHARLES I. WARFIELD

It has been noted fairly consistently that an acceleration of the pulse rate which appears to be disproportionately high has proved to be quite helpful in strengthening the presumptive diagnosis of acute appendicitis complicating pregnancy. This seems to hold true in spite of the temperature, the sedimentation rate, or the leukocytosis.

DIFFERENTIAL DIAGNOSIS

However confusing or difficult the differential diagnosis of acute appendicitis complicating pregnancy may be, two important concepts should be kept in mind: (1) that the mortality of appendicitis is the mortality of delay; and (2) that if it is an appendicitis and it goes on to rupture, it will be followed by a diffuse, spreading peritonitis with little if any tendency to localization or abscess formation.

If these concepts are kept in mind, the examiner will have less tendency to procrastinate in order to obtain an "iron-clad" preoperative diagnosis.

Predilection of right-sided pyelonephritis and hydroureter complicating pregnancy assumes added significance after the twenty-fourth week

TABLE 1
COMMONLY ENCOUNTERED CONDITIONS SIMULATING APPENDICITIS

DISEASES PRODUCING PERITONITIS *Operation Imperative*	DISEASES NOT PRODUCING PERITONITIS *Operation Advisable*	DISEASES NOT PRODUCING PERITONITIS *Operation Contraindicated*
1. Perforated peptic ulcer	1. Intussusception	1. Pyelonephritis
2. Perforated diverticulum of the sigmoid	2. Acute pancreatic necrosis	2. Acute gastroenteritis
3. Ulcerated or perforated Meckel's diverticulum	3. Acute mesenteric thrombosis	3. Acute salpingitis or exacerbation of chronic pelvic inflammatory disease
4. Twisted ovarian cyst		4. Acute cholecystitis
5. Intestinal obstruction		5. Pneumonia and other upper respiratory diseases
		6. Rupture of Graffian follicle
		7. Rotation of uterus
		8. Constipation
		9. Coronary thrombosis and occlusion
		10. Renal colic

Adapted from Cole and Elman: "Text Book of General Surgery," Appleton-Century-Crofts, Inc.

of gestation, as does physiologic rotation of the uterus on its long axis.

Nausea and vomiting which are so common during pregnancy are difficult to evaluate in relation to what may appear to be peritoneal irritation.

Constipation (or with the occasional patient, diarrhea) and the habitual use of laxatives during pregnancy make accurate evaluation of the activity of the gastrointestinal tract very unreliable.

In the latter weeks of gestation in patients with obesity, a large infant, multiple pregnancies, or polyhydramnios abdominal muscle spasm or rigidity is very difficult to ascertain accurately.

Since the symptoms of many surgical and medical conditions often simulate those of acute appendicitis the problem of differential diagnosis becomes one of marked importance. The most commonly encountered conditions whose symptoms often simulate those of appendicitis have been grouped as to peritoneal irritation and operative indication (Table 1).

TREATMENT

MANAGEMENT of this problem, especially during the last trimester of pregnancy and labor, has been the subject of considerable divergence of opinion. It is now generally agreed that once the diagnosis of acute appendicitis complicating pregnancy is made or strongly suspected, immediate operation is indicated. A right rectus incision is preferable since it gives better exposure and facilitates the rapidity of the surgery.

There are those (Cosgrove, McDonald. and TeLinde) who believe that acute appendicitis, with or without rupture, is a surgical condition calling for immediate operative intervention, but should *not* be combined with any manipulation to terminate the pregnancy. Wilson, Zander, Greenhill, and Meiling suggest a Porro cesarean section in the presence of an abscess or perforation. Stoeckel advocates simultaneous appendectomy and vaginal hysterectomy. Lackman and Tusky have suggested the administration of progesterone following surgery during pregnancy.

In view of the experience obtained in the management of acute appendicitis complicating pregnancy the following regimen of therapy which was instituted by the author in the 5 reported cases is suggested:

1. Immediate appendectomy under spinal anesthesia with no attempt to terminate the pregnancy in any way.
2. Penicillin (200,000 units) instilled into the peritoneal cavity. In addition, 1 gm. of streptomycin may be instilled.
3. Peritoneum and abdominal wall closed without drainage.
4. Wangensteen suction in cases of perforation or abscess and p.r.n. in cases with undue postoperative distention.

5. Massive doses of both penicillin and streptomycin parenterally postoperatively.
6. Blood, plasma, and intravenous fluids together with large doses of vitamins B (complex) and C.
7. Stilbestrol 35 mg. and progesterone 10 mg. daily until discharge. The stilbestrol to be continued until delivery to reduce the possibility of abortion or premature labor.
8. As early ambulation as is commensurable with the patient's condition.

MATERNAL MORTALITY

After perforation of the appendix, the mortality rate quoted by most authors is 50 per cent. Kreig reports 7.7 per cent and Meiling 7.69 per cent mortality in the "acute group." Breaking the percentage down still further, McDonald reports a 0.71 per cent maternal mortality when the pathology is limited to the appendix; 23.5 per cent in cases with abscess formation, and 30.0 per cent in those patients developing peritonitis.

FETAL MORTALITY

The incidence of abortion and premature labor increases in exact ratio to the seriousness of the appendiceal inflammation. Meiling's fetal and neonatal mortality rate (including abortions) was 34.6 per cent. McDonald's abortion and fetal mortality increased according to the ratio, with 11.4 per cent when the disease was limited to the appendix and 72.0 per cent with generalized peritonitis. Lackman and Tulsky's abortion rate using progesterone postoperatively was 5.05 per cent.

CASE REPORTS

Case 1 was admitted to the hospital on October 12, 1947, in her thirty-eighth week of gestation. Prenatal course was uneventful. There was a history of abdominal pain about the umbilicus with nausea since that morning, one bout of vomiting previous to admission, and constipation. There was no previous history of abdominal pain.

Physical examination revealed a female in her thirty-eighth week of gestation with the fundus three fingers below the xiphoid; her temperature was 99.2° F., and pulse 120 beats per minute. Pain and tenderness and marked rebound tenderness were noted high in the flank, along with moderate muscle spasm and rigidity. Urinalysis was negative. White blood count was 28,000 with marked shift to the left. A diagnosis of acute appendicitis was made.

At operation, frank purulent exudate was released immediately upon opening the peritoneal cavity with the odor of Escherichia coli. The cecum was adherent to the posterior peritoneum and the surrounding viscera. Only friable particles remained as the stump of the appendix. The remainder of the friable necrotic appendix was retrocecal. It was left intact and a purse-string of cotton suture was placed about the stump and treated with phenol and alcohol. One gram of streptomycin and 200,000 units of penicillin were instilled in the peritoneal cavity. The peritoneum and abdominal wall were closed in layers without drainage. The patient was placed on the postoperative regimen described. She was discharged on the fourteenth postoperative day.

She returned to the hospital on the nineteenth postoperative day and delivered a living infant without difficulty per vaginam.

Case 2 was admitted to the hospital on November 27, 1947, in her thirty-second week of gestation. Her prenatal course was uneventful. There was a history of acute onset of abdominal pain which radiated into the right flank, and nausea, vomiting, and diarrhea since the previous afternoon.

Physical examination revealed a female in the thirty-second week of gestation, with a temperature of 99° F., pulse 100 per minute. The pain and tenderness were severe enough to compel the patient to draw her legs against her abdomen. There was moderate rebound tenderness; no rigidity, but slight to moderate spasticity in the right flank about half way between the iliac crest and the costal margin. Urinalysis was essentially negative. White blood count was 12,500 with shift to the left. The diagnosis was acute appendicitis.

At operation, the appendix was found to be retrocecal, subserous, and 15 cm. in length. The base was located and ligated and the appendix was removed in a retrograde manner. The appendix was swollen, injected, and gangrenous in the distal 2 cm. One gram of streptomycin and 200,000 units of penicillin were instilled in the peritoneal cavity. The peritoneum and abdominal wall were closed without drainage.

The pathologic diagnosis was acute gangrenous appendicitis without perforation. The patient was placed on the postoperative regimen described, and was discharged on the tenth postoperative day. She returned at term and delivered a living infant per vaginam uneventfully.

Case 3 was admitted to the hospital on November 17, 1947, in her tenth week of gestation, with a history of vague abdominal cramps about the umbilicus and epigastrium, progressing to the right lower quadrant, of approximately eight hours' duration. There was constipation, some nausea, but no vomiting.

Physical examination revealed a female in her tenth week of gestation, with a temperature of 98.6° F., and a pulse of 90 per minute. There was moderate pain and tenderness in the right lower quadrant with slight rebound tenderness and moderate voluntary rigidity. Urinalysis was negative; white blood count was 8,500 with slight shift to the left.

She was given 50,000 units of penicillin parenterally every three hours and supportive therapy was instituted. She failed to improve after six hours. A diagnosis of acute appendicitis was made.

At operation, the appendix was found to be moderately edematous and injected. It was removed and 1 gm. of streptomycin and 200,000 units of penicillin were instilled in the peritoneal cavity. The peritoneum and abdominal wall were closed without drainage.

The pathologic diagnosis was acute appendicitis. The patient was placed on the postoperative regimen described, and was discharged on the tenth postoperative day. She returned at term and delivered a living infant per vaginam uneventfully.

Case 4 was admitted to the hospital on February 28, 1947, in her twenty-fourth week of gestation, with a history of progressive abdominal pain for twelve hours, and nausea and vomiting for five hours. There was no change in normal bowel habits; she had had a bowel movement that morning.

Physical examination revealed a female in her twenty-fourth week of gestation, with a temperature of 100° F., and pulse of 90 beats per minute. There was severe pain, tenderness, and rebound tenderness in the right lower quadrant and right flank, with moderate rigidity on the right. Urinalysis showed one plus sugar and a trace of albumin, but otherwise was negative. Sedimentation rate was moderately elevated. White blood count was 15,200 with definite shift to the left. A diagnosis of acute appendicitis was made.

At operation, the appendix was found to be edematous and markedly injected. It was removed, and 1 gm. of streptomycin and 200,000 units of penicillin were instilled in the peritoneal cavity. The peritoneum and abdominal wall were closed without drainage.

The pathologic diagnosis was acute suppurative appendicitis. The patient was placed on the postoperative regimen described, and was discharged on the tenth postoperative day.

Spontaneous labor with rupture of the membranes ensued three weeks after discharge. She was delivered by manual rotation of posterior position and easy midforceps after the infant's head failed to advance beyond the midpelvis. The infant's weight was 4 pounds, 15 ounces. Both mother and infant were discharged on the ninth postpartum day.

Case 5 was admitted to the hospital on March 6, 1947, in her twentieth week of gestation, with a classical history of acute appendicitis—nausea, vomiting, constipation, progressive abdominal pain beginning about the umbilical region and migrating to the right side of the abdomen.

Physical examination revealed a female in her twentieth week of gestation, with a temperature of 99° F., and pulse of 112 beats per minute. There was moderately severe abdominal pain confined to the right side and right flank, with moderate rebound tenderness and some suggestive spasticity on the right. Urinalysis was negative. Sedimentation rate was elevated to 30 mm. in one hour. White blood count was 15,000 with a definite shift to the right. A diagnosis of acute appendicitis was made.

At operation the appendix was found to be acutely inflamed with a large, hard fecalith in the center, entirely occluding the lumen. Penicillin (200,000 units) and streptomycin (1 gm.) were instilled in the peritoneal cavity. The peritoneum and abdominal wall were closed without drainage.

The pathologic diagnosis was acute appendicitis. The patient was placed on the postoperative regimen described, and was discharged on the tenth postoperative day. She returned at term and delivered a living infant spontaneously per vaginam.

The reported cases are from the Obstetric and Gynecologic Service, U. S. Air Force Hospital, MacDill, Tampa, Florida (1947).

I wish to thank Dr. O. A. Ellingson for his cooperation in these cases; and Drs. A. A. Marchetti and C. F. Geschickter for their invaluable advice during the preparation of this report.

REFERENCES

1. Cole, W. H., and Elman, R.: Text Book of General Surgery. New York, Appleton-Century-Crofts, Inc., 1936.
2. Cosgrove, S. A.: Surgical complications of pregnancy. Am. J. Obst. & Gynec. 34:169-179, 1937.
3. Crossen, H. S., and Crossen, R. J.: Operative Gynecology. Ed. 6. St. Louis, C. V. Mosby Company, 1948.
4. DeLee, J. B., and Greenhill, J. P.: Principles and Practices of Obstetrics. Ed. 9. Philadelphia, W. B. Saunders Company, 1947.
5. DeVoe, R. W., Day, L. A., and Ferris, D. O.: Pregnancy at term complicated by ruptured appendix. Proc. Staff Meet., Mayo Clinic 22:135-141 (April 2) 1947.
6. Fegtly, A. W.: Appendicitis associated with pregnancy. J. Kansas M. Soc. 39:508-513 (December) 1938.
7. Fwyman, R. A., Mussey, R. D., and Stalker, L. K.: Appendicitis in pregnancy; review of 75 cases. Proc. Staff Meet., Mayo Clin. 15:484-487 (July 31) 1940.
8. Garmon, I. A.: Acute appendicitis with case report. Virginia M. Monthly 74:115-117 (March) 1947.
9. Hoffman, E. S., and Suzuki, M.: A comparative study of acute and chronic appendicitis in pregnancy. West. J. Surg. 57:150-157, 1949.
10. Hoffman, E. S., and Suzuki, M.: Acute appendicitis in pregnancy. Grace Hosp. Bull. 26:25-46, 1948.
11. Jarvis, C.: Pregnancy complicated by acute appendicitis. Harper Hosp. Bull. 5:85-86 (June) 1947.
12. Kreig, E. G. M.: Appendicitis occurring during pregnancy: A diagnostic review of 200 cases. Am. J. Obst. & Gynec. 57:736-741, 1949.
13. Lackner, J. E., and Tulsky, A. S.: Abortion as a complication of operation on the pregnant woman. Am. J. Surg. 46:362-364, 1939.
14. Long, I. P.: Appendicitis complicating pregnancy. Memphis M. J. 22:36-38 (March) 1947.
15. Meiling, R. L.: Appendicitis complicating pregnancy, labor and puerperium. Surg., Gynec. & Obst. 85:512-522 (October) 1947.
16. Milnor, G. C.: Acute suppurative appendicitis with diffuse peritonitis at fourth month of pregnancy—case. Proc. Staff Meet., Clin. Honolulu 8:41-42 (June) 1942.
17. McGehee, J. L.: Acute appendicitis in pregnancy. Mississippi Doctor 19:238-241 (October) 1941.
18. Norton, J. F., and Connell, J. N.: Appendicitis complicating pregnancy and labor. Am. J. Surg. 32:325-332, 1936.
19. Potter, M. G., and Saducor, M. G.: Term pregnancy complicated by a ruptured appendix with generalized peritonitis. West. J. Surg. 57:120-122, 1949.
20. TeLinde, R. W.: Operative Gynecology. Philadelphia, J. B. Lippincott Co., 1946.
21. Turner, S. J., and Weiss, M. B.: Acute suppurative appendicitis and pregnancy at term. Am. J. Obst. & Gynec. 56:1003-1005, 1948.
22. Underwood, J H.: Appendicitis complicating pregnancy. J. M. Soc., New Jersey 40:62-63 (February) 1943.
23. Zander, E. L.: Acute appendicitis as a complication of pregnancy. West. J. Surg. 50:360-364 (July) 1942.

TUBERCULIN NEGATIVE, HISTOPLASMIN POSITIVE
Disseminated Pulmonary Calcification

MICHAEL L. FURCOLOW*

UNITED STATES PUBLIC HEALTH SERVICE, KANSAS CITY, KANSAS

DISSEMINATED pulmonary calcification has been the subject of considerable interest in the literature of recent years. Originally considered the end result of the healing of miliary or disseminated tuberculosis,[1-5] it was nevertheless suspected to be of fungus etiology by Sayers and Meriwether[6] and Spolyar.[7] The rather peculiar geographic limitation to the east-central United States was noted by Long and Stearns.[8]

With the finding of a definite correlation between pulmonary calcification and reaction to histoplasmin (an extract of the fungus *Histoplasma capsulatum*) by Palmer[9] and Christie and Petersen,[10] more concrete relationship between fungus infection and calcification began to emerge. Zwerling and Palmer[11] reported 15 cases of disseminated calcification, 14 of which reacted to histoplasmin while High, Zwerling, and Furcolow[12] reported 108 cases of which 104 reacted to histoplasmin, none to tuberculin only, and 11 to both tuberculin and histoplasmin. The present paper will serve to bring up to date this earlier paper and present illustrative x-rays of the development of disseminated pulmonary calcification in patients under observation.

In addition to the reports cited, the author has been privileged to review a paper by White and Hill[13] which is now in press. This paper presents the results of histoplasmin and tuberculin skin tests on 114 cases of disseminated calcification as well as detailed histories and x-rays on a number of these cases followed from the onset of the disseminated pulmonary disease to final calcification some years later. Ninety-four per cent of their cases reacted to histoplasmin and 50 per cent to tuberculin and histoplasmin, while only 2 per cent reacted to tuberculin only. In addition to skin tests, histoplasmosis complement fixation tests employing yeast phase antigen were performed in 9 individuals. Eight were found to have a positive and one a doubtful reaction to this test. Of particular interest is the correlation of the disseminated pulmonary calcification with histoplasmin sensitivity in an area (upper New York state) where the histoplasmin sensitivity of the general population is low (10 to 14 per cent).

*Senior Surgeon, United States Public Health Service; From the Division of Tuberculosis, Field Studies Branch, University of Kansas Medical Center, Kansas City, Kansas.

Presented November 1, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

MATERIAL AND METHODS

Details of collection of this material were given in the previous paper.[12] Briefly, a total of 15,980 children were given tuberculin and histoplasmin tests and were examined with an 11″ by 14″ or 14″ by 17″ roentgenogram of the chest. Among the roentgenograms of these approximately 16,000 children, 62 instances of disseminated pulmonary calcification were noted. The tuberculin used in these studies was 0.0001 mg. of PPD-S, furnished by Dr. Florence Seibert of the Henry Phipps Institute, University of Pennsylvania, Philadelphia; the histoplasmin was a 1:1000 dilution of lots H-3 or H-15 furnished by Dr. C. W. Emmons or Dr. Arden Howell, Jr., of the United States Public Health Service. All injections were made intradermally. A reaction was considered positive if the in-

MICHAEL L. FURCOLOW

duration measured 5 mm. or more in diameter at the forty-eight-hour reading.

The cases of disseminated pulmonary calcification to be presented in this paper have been restricted to those instances in which at least five separate calcareous deposits were noted in each lung field. Further, the deposits must have been scattered over at least one-half of each lung field. In almost every instance, these minimal requirements were exceeded. Through adherence to these criteria, disseminated pulmonary calcifications may be divided into two groups. The first is designated "miliary calcification" (Figures 1 to 8). The calcifications are small, round, uniform in size, numerous, and widely and symmetrically scattered throughout each lung field. This type is sometimes called "wheatena" or "buckshot" calcification. The second group is designated "multiple bilateral calcification" (Figure 9). In these instances the calcareous deposits are fewer in number, often irregular in outline, of varying size, and often distributed in an asymmetric pattern. In each group, two subgroups may be made, according to whether calcareous deposits are observed in the hilar regions.

The distinction between the two groups is of interest because most observers feel that the "miliary" type results from hematogenous dissemination of the causative agent,[1-5] whereas bronchogenic dissemination produces the "multiple bilateral" type.[5]

FINDINGS

In Figures 1 to 8 are presented reproductions of x-rays of 4 cases of miliary pulmonary calcification. Two x-rays from each case are presented with a time interval of two to four years between them. The development of calcification in the miliary lung lesions is clearly illustrated. The first 2 cases for which x-rays are shown (Figures 1 to 4) are proved cases of histoplasmosis from which the organism was recovered by culture.[14]

Figure 9 illustrates what has been called the multiple bilateral type of pulmonary calcification. The irregular size and lack of uniform distribution throughout the lung fields are clearly evident.

In Table 1 are presented the distribution of cases of disseminated pulmonary calcification according to age and race.

It is apparent that there were not sufficient cases among the Negro children for age rates to be computed. However, the over-all prevalence among the Negroes was only 1.2 per thousand as compared with 4.5 per thousand among the white children. This racial difference is statistically significant and was previously noted by Lumsden and Dearing.[15] There is a steady rise in rates among the white children from 0 per 1,000 persons in the youngest age group to 10 per 1,000 among the age group of 16 to 18 years.

The differences in prevalence of disseminated pulmonary calcification between white males and females were found not to be statistically significant. However, there was a slightly greater frequency among females than among males.

It should be stated that all types of pulmonary calcification observed among these Kansas City school children were more frequently found in histoplasmin reactors than in tuberculin reactors. Furcolow, et al.[16] studied 6,528 school children who were part of the same group in which the cases of disseminated pulmonary calcification were found. They included only those children, however, whose chest roentgenograms were entirely satisfactory for interpretation of all types of calcification. The present report deals with the entire group because it is felt that dissemi-

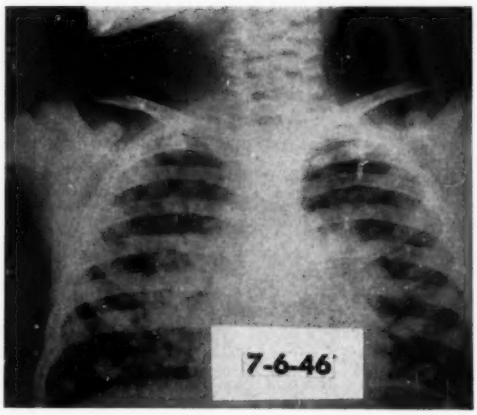


FIGURE 1. Case 1, x-ray taken 7-6-46 of a 3-year-old boy, a proved case of histoplasmosis.[14] Disseminated soft infiltrates throughout both lungs with bilateral enlargement of the lymph nodes.

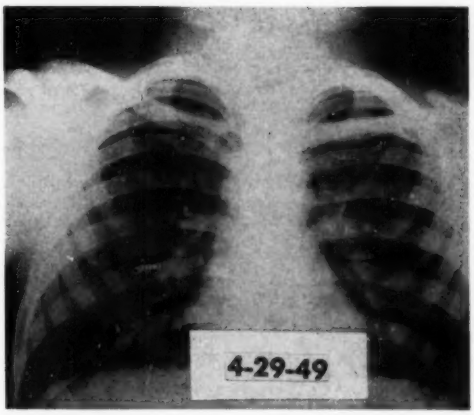


FIGURE 2. Case 1, x-ray taken 4-29-49 showing that miliary small calcification has developed throughout both lungs.

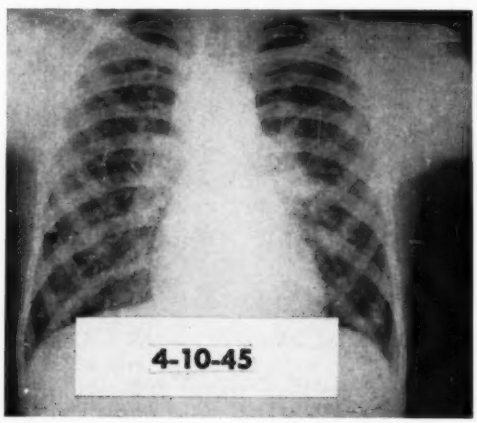


FIGURE 3. Case 2, x-ray taken 4-10-45 of a 13-year-old boy, a proved case of histoplasmosis.[14] Same x-ray appearance as Figure 1.

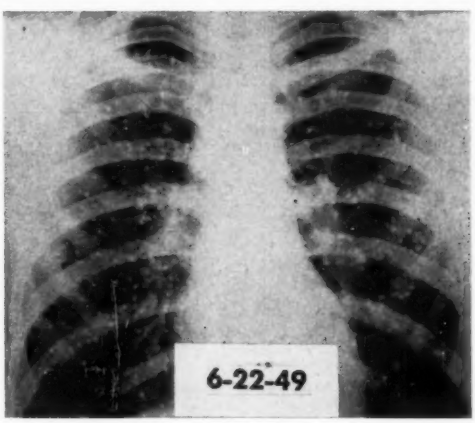


FIGURE 4. Case 2, x-ray of 6-22-49 showing the development of miliary calcified nodules throughout both lung fields.

nated calcification would be seen even on films of poor technical quality.

Among the 6,528 school children studied,[16] 828 cases of pulmonary calcification were found. Of the 828 cases, 56, or 6.8 per cent, occurred among children positive to both skin tests. Among those who reacted only to histoplasmin, 649 cases, or 78.4 per cent, were found. Thirty-one cases, or 3.7 per cent, were found among those who reacted only to tuberculin; and 92, or 11.1 per cent were found among those who reacted to neither test. In Table 2, these results are compared with those found among the children with disseminated calcification.

It is important to note that the percentage of histoplasmin reactors was higher among those with disseminated calcification than among those with all types of pulmonary calcification. Among the former, 96.7 per cent reacted to histoplasmin (or to histoplasmin and to tuberculin); and among the latter, only 85.2 per cent reacted. The difference is significant.

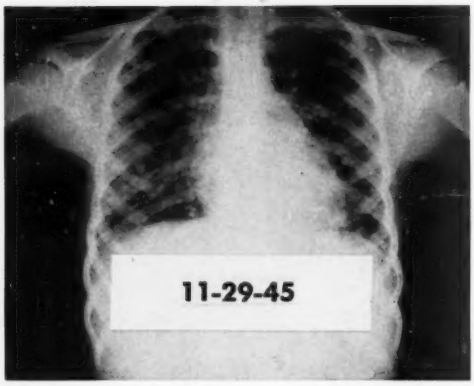


FIGURE 5. Case 3, x-ray taken 11-29-45 on a 6-year-old girl. Disseminated nodular fairly hard infiltrations throughout both lung fields.

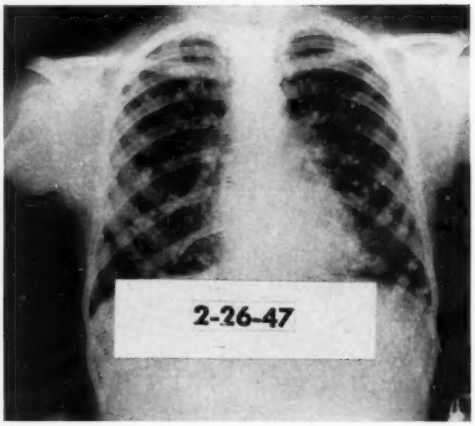


FIGURE 6. Case 3, x-ray of 2-26-47 showing the development of calcification in the lesions.

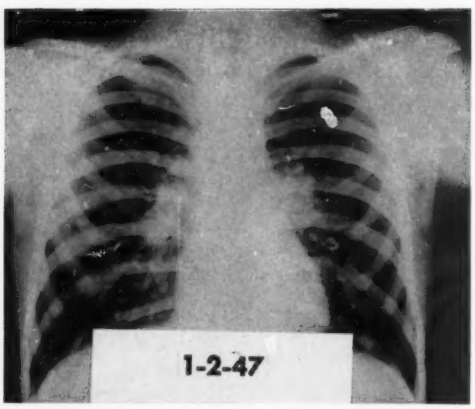


FIGURE 7. Case 4, x-ray taken 1-2-47 on a 9-year-old boy showing many soft infiltrations scattered throughout both lungs and bilateral enlargement of the lymph nodes.

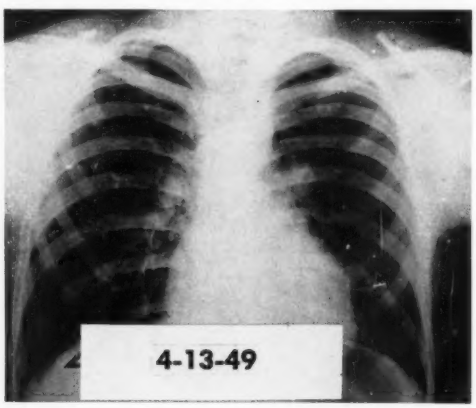


FIGURE 8. Case 4, x-ray of 4-13-49 showing the development of fine miliary calcification.

From the above findings, it appears that tuberculosis, contrary to the opinion of many previous writers, is not the cause of the majority of such calcifications. Among the 62 cases, 60, or 96.7 per cent, did not react to tuberculin, and only 2 or 3.2 per cent, reacted to tuberculin as well as to histoplasmin.

In the previous paper[12] a total of 113 cases of disseminated pulmonary calcification was collected from all sources, including school children, adults, and student nurses. These 113 cases were collected from among approximately 44,000 persons examined up to that time. Since this report was submitted, approximately 16,000 additional adults and children have been examined in the Kansas City area. Among this group an additional 66 cases of disseminated pulmonary calcification have been found. The total of cases collected to date is, therefore, 174 found among approximately 60,000 persons. This total excludes 5 cases of the original 113 who were not tested with tuberculin and histoplasmin. The results of tuberculin and histoplasmin skin tests on the 174 cases are presented in Table 3.

One case (0.6 per cent) was found with a positive tuberculin reaction alone, whereas 86.0

TABLE 1

Cases of Disseminated Pulmonary Calcification Per 1,000 Persons by Age and Race—School Children, Kansas City, Missouri, 1945

Age[1] (years)	White and Negro			White			Negro		
	Number		Rate per 1,000 persons tested	Number		Rate per 1,000 persons tested	Number		Rate per 1,000 persons tested
	Children	Cases		Children	Cases		Children	Cases	
0-4	242	0	0	207	0	0	35	0	0
4-6	2,482	2	.8	2,084	2	1.0	398	0	0
7-9	3,594	9	2.5	2,857	7	2.5	737	2	2.7
10-12	3,628	14	3.9	2,868	14	4.9	760	0	0
13-15	3,966	21	5.3	3,679	20	5.4	287	1	3.5
16-18	2,068	18	8.7	1,802	18	10.0	266	0	0
Total	15,980	64	4.0	13,497	61	4.5	2,483	3	1.2

[1]Age last birthday.

TABLE 2

Percentage Distribution by Reactions to Histoplasmin and Tuberculin for All Children Tested—For Children with Pulmonary Calcification and for Children with Disseminated Calcification—School Children, Kansas City, Missouri, 1945

Skin reaction	Among 6,528 School Children[1]				Disseminated calcification among 15,980 school children	
	All children		Children with all types of pulmonary calcification		Total	Percentage
	Total	Percentage	Total	Percentage		
H+ T−	2,454	37.6	649	78.4	58	93.5
H+ T+	235	3.6	56	6.8	2	3.2
H− T+	273	4.2	31	3.7	0	0
H− T−	3,566	54.6	92	11.1	2	3.2
Total	6,528	100.0	828	100.0	62	100.0

[1]From Furcolow, et al.[10]

TABLE 3

Cases of Disseminated Pulmonary Calcification Collected from All Sources, According to Reaction to Histoplasmin and Tuberculin

Skin reaction	Total	Multiple bilateral type		Miliary type	
		With hilar calcification	With no hilar calcification	With hilar calcification	With no hilar calcification
H+ T−	149	66	18	39	26
H+ T+	21	10	1	7	3
H− T+	1	1	0	0	0
H− T−	3	0	1	1	1
Total	174	77	20	47	30

per cent reacted to histoplasmin alone. Of the 174 cases, 1.7 per cent reacted to neither skin test, and 12.0 per cent reacted to both. Therefore, 98.0 per cent of the cases showing this type of calcification reacted to histoplasmin, and only 13 per cent reacted to tuberculin.

Of the total group of 174 cases, 97, or 55.7 per cent, were of the multiple bilateral type; while 77 or 44.3 per cent, were of the miliary type. Calcifications were noted in the hilar structures in 79.4 per cent of the former type, whereas only 61.0 per cent of the latter type showed such calcifications. No significant differences were observed in the skin reactions to histoplasmin or tuberculin in these two groups, regardless of the presence or absence of calcifications in the hilar areas.

Complement fixation tests for histoplasmosis[17] were performed on the blood serum of 42 of these 174 cases. Of the 42 tested, 13, or 31 per cent, were positive (++++); 11, or 26 per cent, were doubtful (+++ or ++); and 18, or 43 per cent, were negative (+ or 0). It should be stated that most of the cases reported herein appeared by x-ray to be healed and that the complement fixation test was not developed until two years after most of them were first observed.

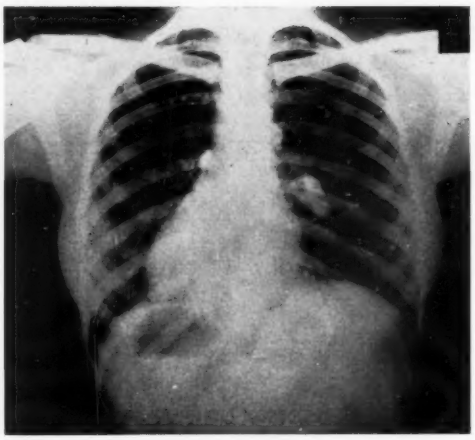


FIGURE 9. Case 5, x-ray showing what is termed multiple bilateral pulmonary calcification with very large calcified hilar lymph nodes.

SUMMARY

ONE hundred and seventy-four instances of disseminated pulmonary calcification are reported, and the skin reactions to tuberculin and histoplasmin are given. Reproductions of x-rays from selected cases show the development of this type of calcification.

Of the 174 cases, 170, or 98.0 per cent, reacted to histoplasmin, while only 4 had negative reactions to this antigen. One case reacted only to tuberculin. From evidence presented in this paper and accumulated from the literature, it appears probable that:

1. Tuberculosis is an exceedingly infrequent cause of disseminated pulmonary calcification.

2. Histoplasmosis or some closely related infection is the cause in most cases. This probability is supported by the cultural isolation of *Histoplasma capsulatum* from 2 cases whose x-rays are reproduced.

Observations of the frequency of the occurrence of disseminated calcification among 16,000 school children indicate that:

1. White children show a frequency of 4.5 per 1,000 while Negroes show only 1.2 per 1,000.

2. Among white children the frequency increases with age from none in the age group under 4 years to 10 per 1,000 in the age group of 16 to 18 years.

REFERENCES

1. OPIE, E. L., and ANDERSEN, H.: First infection with tuberculosis by way of the lungs. Am. Rev. Tuberc. 4:629-640, 1920.
2. MIDDLETON, W. S.: Healed miliary tuberculosis of the lung. Am. J. Roentgenol. 14:218-221, 1925.
3. BAER, R. W.: Report of a case of healed miliary tuberculosis. Am. J. Dis. Child. 27:110-112, 1924.
4. PIERSON, P. H.: Healed generalized miliary tuberculosis. Am. Rev. Tuberc. 13:342-349, 1926.
5. MAYORAL, A.: Multiple miliary calcifications in the lung. Radiology 36:367-371, 1941.
6. SAYERS, R. R., and MERIWETHER, F. V.: Miliary lung disease due to unknown cause. Am. J. Roentgenol. 27:337-351, 1932.
7. SPOLYAR, L. W.: Undiagnosed pulmonary miliary calcifications in a group of war workers. Quart. Bull. Ind. Univ. Med. Center 6:3-6, 1944.
8. LONG, E. R., and STEARNS, W. H.: Physical examination at induction. Radiology 41:144-150, 1943.
9. PALMER, C. E.: Nontuberculous pulmonary calcification and sensitivity to histoplasmin. Pub. Health Rep. 60:513-520 (May) 1945.
10. CHRISTIE, AMOS, and PETERSEN, J. C.: Pulmonary calcification in negative reactors to tuberculin. Am. J. Pub. Health 35:1131, 1945.
11. ZWERLING, H. B., and PALMER, C. E.: Pulmonary calcification: Roentgenographic observations in relation to histoplasmin and tuberculin reactions. Radiology 47:59-63, 1946.
12. HIGH, ROBERT H., ZWERLING, HENRY B., and FURCOLOW, MICHAEL L.: Disseminated pulmonary calcification. Pub. Health Rep. 62:20-29 (January) 1947.
13. WHITE, F. CLARK, and HILL, HARRY E.: Disseminated pulmonary calcification: A report of 114 cases with observations of an antecedent pulmonary disease in fifteen individuals. Am. Rev. Tuberc. (In press.)
14. FURCOLOW, MICHAEL L.: (Paper in preparation.)
15. LUMSDEN, L. L., and DEARING, W. P.: Epidemiological studies of tuberculosis. Am. J. Pub. Health 30:219-228, 1940.
16. FURCOLOW, M. L., HIGH, R. H., and ALLEN, M. F.: Some aspects of sensitivity to histoplasmin and tuberculin. Pub. Health Rep. 61:1132-1144 (August) 1946.
17. FURCOLOW, M. L., BUNNELL, I. L., and TENENBERG, D. J.: A complement fixation test for histoplasmosis. Pub. Health Rep. 63:169-173 (February) 1948.

Diagnostic Clinic

Medical Management of Thyroid Disease

MICHAEL G. WOHL*

TEMPLE UNIVERSITY SCHOOL OF MEDICINE, PHILADELPHIA

When one follows the cycle of a patient with hyperthyroidism one is impressed by the fact that this disorder is a widespread disease of the neuroglandular system with many manifestations of an overactive thyroid gland.

The cause of the disease is still unknown, but apparently does not reside within the thyroid gland. Furthermore, one is forced to conclude that the removal of a major part of the thyroid gland either by surgical means or by the modern antithyroid drugs brings about a remission, and if that remission lasts long enough, a year or longer, the disease may subside spontaneously. One cannot speak of a cure of the disease, for we all know of cases that have had long remissions—ten or fifteen years after surgery; then a recurrence took place which required a second and sometimes even a third and fourth surgical intervention. Whether the same results will be obtained in patients after prolonged antithyroid medication, no one knows. The life cycle of patients who have been treated by antithyroid drugs has not been followed long enough to know that.

I shall discuss the clinical course of some patients who have been under prolonged medication with antithyroid drugs plus iodine, and try to illustrate some of the problems involved in managing these patients. Then Dr. Robbins will follow with a discussion of radioactive iodine.

In order to clarify my remarks, I should like briefly to review the modern concepts of thyroid physiology.

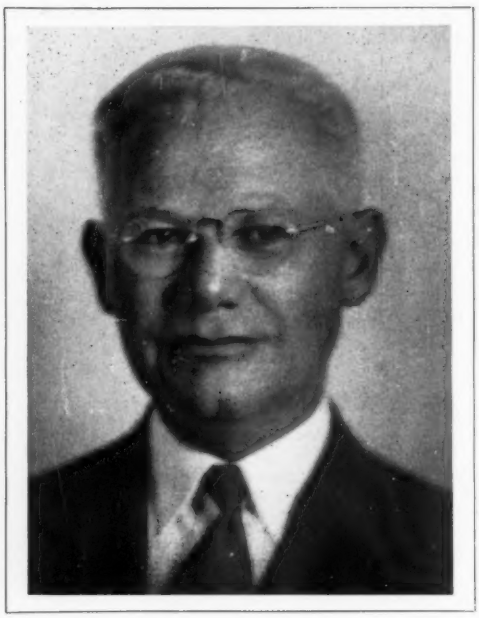
The thyroid gland concentrates and traps inorganic iodine derived from food or from broken down tissue, and it is within the follicles by means of an enzyme system that free iodine is liberated and unites with protein to form two moieties, (1) diiodotyrosine, which possesses no hormone activity but is merely a chemical stepping-stone toward the formation of the active thyroid principle; (2) thyroxin, which diffuses from the follicle into the circulation and is carried to the peripheral tissues of the body.

The output and secretion of the thyroid hormone is under direct influence of the TSH, the thyrotropic hormone of the pituitary gland. From the circulation it is carried into the thyroid where it stimulates the thyroid gland, causing it to undergo hyperplasia, and releases thyroxin, which is carried to the tissues and is responsible for the basal oxidation processes. This TH, carried back to the pituitary, in its turn inhibits output of TSH, so there is a reciprocal regulatory relationship established whereby one gland controls the other.

In hyperthyroidism the anterior pituitary plays an important role. It either secretes TSH in excessive amounts in a gland that is normally sensitive or it secretes a normal amount of TSH in the thyroid gland which is hypersensitive. It is important to bear in mind that the antithyroid drugs act by inhibiting the peroxidase enzyme system, whereby free iodine is liberated from

*Associate Professor of Medicine and Chief of Endocrine Clinic, Temple University School of Medicine: Chief of Nutrition Clinic, Philadelphia General Hospital, Philadelphia, Pennsylvania.

Presented November 3, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.



MICHAEL G. WOHL

iodide; in this way less iodine is available for thyroxin formation, and therefore no TH is carried to the body tissue (Figure 1). Hence the basal metabolic rate falls, the organic iodine concentration is decreased in the blood and as well in the thyroid gland, and the patient to all intents and purposes shows marked improvement, but there is a paradoxical histologic occurrence, and that is this: In spite of the fact that the patient is clinically very much improved and from a laboratory angle shows a fall in the basal metabolic rate and protein-bound iodine, the thyroid gland shows hyperplasia similar to that seen in the active stage of the disease. The reason for this is there is no thyroxin carried to the pituitary; therefore the brakes are, so to speak, off, and there is a constant stimulation and hyperplasia of the thyroid without the formation of thyroxin.

When you add Lugol's solution in the presence of antithyroid drugs, the thyroid gland does not lose its capacity to store inorganic iodine and therefore the iodine inactivates the TSH at the level of the thyroid so that the cells in the follicles undergo resting or regressive changes; deposition of colloid takes place and involution is said to occur. The gland then becomes firm instead of vascular as before and is less friable, and there is very little thyroxin secreted because in addition to this action upon TSH at the thyroid level it also acts upon the proteolytic enzymes within the follicle and thus further inhibits thyroxin formation. From this sketchy review it becomes understandable why the combined use of iodine and antithyroid drugs like propyl thiouracil has a more beneficial effect therapeutically; it hastens the remission and the magnitude of improvement is far greater than when antithyroid drugs are used alone (Figure 2).

THE medical management of hyperthyroidism consists in the administration of antithyroid drugs. There are many on the market and that most commonly used in this country today is propyl thiouracil; lately methyl thiouracil is coming into use. Methyl thiouracil has been more extensively used in Australia and Europe. Propyl thiouracil is given in doses of 150 to 200 mg. a day, in more severe cases 200 to 300 mg., in combination with Lugol's solution 5 to 15 minims daily. These drugs are given for a period of three to four months, when a real improvement takes place, and then the dosage may be cut down according to the patient's condition. When the basal metabolic rate is reduced to 0, propyl thiouracil and iodine are continued in reduced amounts for a period of ten months—preferably twelve months—and at the end of this time the drugs are stopped with the expectation that a lasting remission will occur.

In addition to these drugs we must not forget that these patients need other care, particularly nutritional care, especially in the active stage of the disease. Hyperthyroid patients lose a lot of nitrogen, they lose a lot of calcium, and they also have a heightened requirement for the various nutrient factors. If these are not supplied in adequate amounts a deficiency syndrome may ensue which may not only complicate but aggravate the condition, and I will show you in a few moments just how this happened to one of our patients.

The first patient had lost about 35 pounds in weight within a period of four months following the death of her husband. She became very fatigued, developed palpitation, showed slight exophthalmos, nervousness, and tachycardia, the typical symptoms of hypermetabolism. Her basal metabolic rate was +38. She was given 300 mg. of propyl thiouracil and Lugol's solution 15

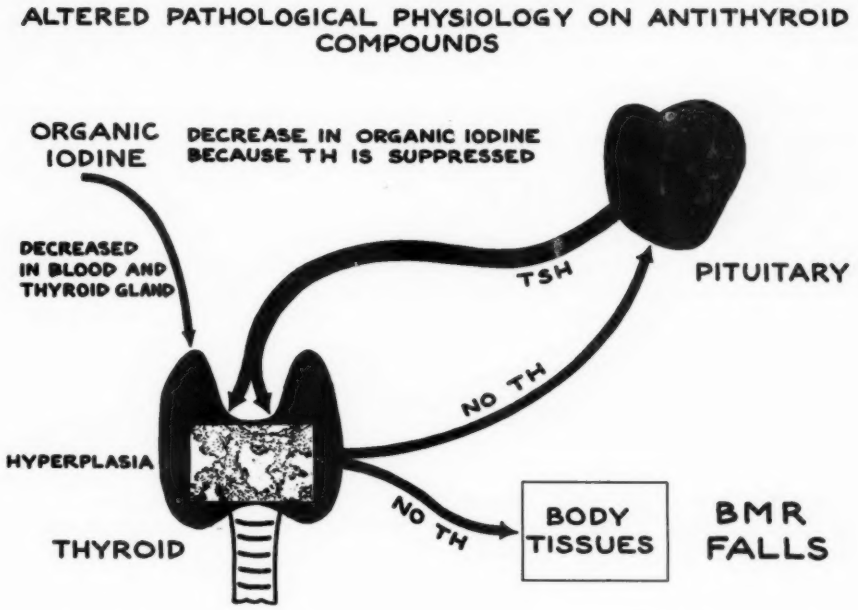


FIGURE 1. Outline of altered pathologic physiology in hyperthyroidism following administration of antithyroid compounds.

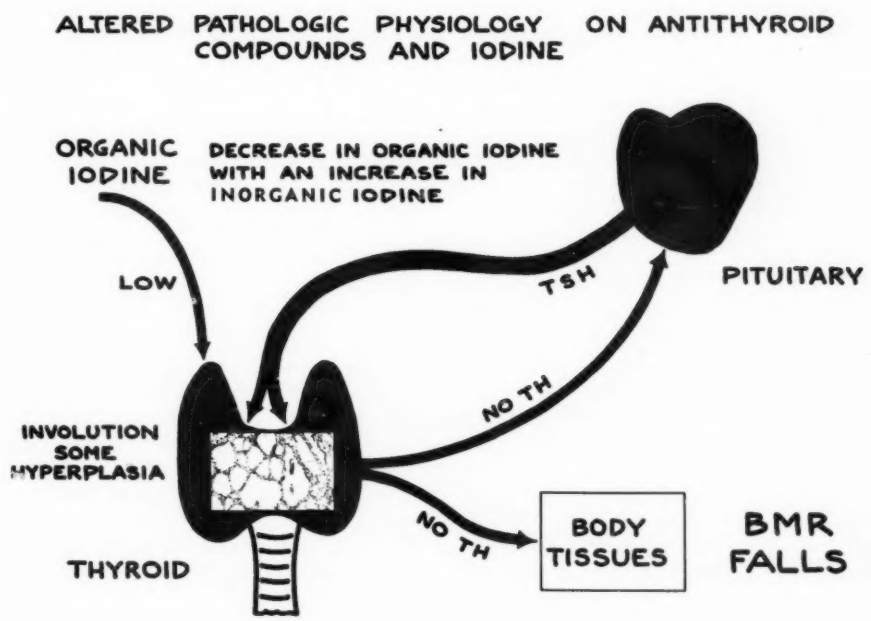


FIGURE 2. Outline of altered pathologic physiology in hyperthyroidism following administration of antithyroid compounds and iodine.

minims daily, and in three months she gained 12 pounds in weight and improved considerably. The dose was reduced to 200 mg. and iodine to 10 minims daily and continued for another two months until the basal metabolic rate came to −5. She was maintained on 100 mg. of propyl thiouracil for a period of seven months. At the end of twelve months her basal metabolic rate came down to −13, her pulse was 70, her exophthalmos somewhat receded, there was no particular swelling of the neck, the thyroid was inconspicuous, and she felt well. All medication was withdrawn. She has been without medication for a period of sixteen months and to all intents and purposes is free from hyperthyroid symptoms.

This patient represents a highly satisfactory result. We have similar cases that had prolonged remissions on the combined treatment of propyl thiouracil and iodine, with remissions lasting for periods of ten to twelve months. How many more would have had remissions is difficult to state because many of these patients have not been faithful enough to stick to the medication and they either drifted away or had surgery.

The next patient has had a long story. She has had hyperthyroidism or, rather, Graves' disease, for a period of ten years, for which she has been given iodine off and on. She refuses surgery. Four months prior to her admission to the Temple University Hospital she stopped taking iodine. She had lost considerable weight, and was very toxic. Her basal metabolic rate was +50, she was fibrillating, had extreme nervousness, and palpitation. In addition to these general hypermetabolic symptoms she also had a condition on her lower extremities which had resisted all forms of dermatologic care. There was a red, angry-looking eczematoid condition of both lower extremities which to us looked like that of pellagra due to the exhaustion of nicotinic acid because of the heightened metabolic rate and insufficient intake of the proper nutrients.

She was put on thiouracil, given proper nutritive care with increased amounts of nicotinic acid and the eczematoid condition completely cleared up and has remained clear.

The patient continued to do well for some time until she developed a leukopenia of about 3,000 cells, 20 per cent polymorphonuclear leukocytes, and the thiouracil was stopped, with no treatment for eight weeks, and the basal metabolic rate rose again. She was put on propyl thiouracil, small amounts of about 100 mg. twice daily with iodine, and in the course of three months she made constitutionally marked improvement, gained considerable weight, but developed a granulocytopenia of 2,000 cells, 15 per cent polymorphonuclears, and had a sore throat with a temperature of 102° F.

We got a little frightened about this because we had a similar experience some years ago with thiouracil that proved fatal. We omitted all medication, gave her penicillin and intramuscular injections of crude liver, and in four days her throat cleared up and the leukocyte count had risen to 6,000 with 44 per cent polymorphonuclear leukocytes. She was then given Lugol's solution and we began to feel our way.

Finally she has been taking 100 mg. of propyl thiouracil daily in combination with Lugol's solution and has been doing very well indeed. She has gained in weight from 114 to 147 pounds and is able to carry on her household duties, and she tells me she was able to take care of a boarding house, doing heavy work, until recently when she had an upper respiratory infection and is again beginning to show some evidence of a recurrence.

I am trying to convince this lady to have something else done besides medication because of the frequent relapses, but she still refuses surgery. Maybe I will send her to Dr. Robbins for radioiodine after she gets through listening to him today.

The third patient has been treated for menopause. She is one of the masked cases. When I saw her she presented symptoms of hyperthyroidism—loss of weight, nervousness, a basal metabolic rate of +37, with a tachycardia of 110. She was placed on propyl thiouracil, 200 mg. in combination with Lugol's solution 15 minims daily, and was doing very well when treatment was stopped after four months and she had a recurrence. We put her back on treatment and she did well. She became a little bit fretful and discouraged by the fact that she had to come for frequent checkups and felt that perhaps it was to be a lifelong treatment, so she asked for surgery. She had subtotal thyroidectomy done and made an uneventful recovery. But about five weeks later she developed numbness and tingling in both hands and the toes of her feet, and the toes became flexed upon walking. She developed postoperative tetany and

TABLE 1

ADVANTAGES AND DISADVANTAGES OF SURGICAL TREATMENT OF HYPERTHYROIDISM

ADVANTAGES
Lasting remission in more than 90 per cent
Pressure symptoms and deformities relieved
DISADVANTAGES
Operative mortality ranges from 0.08 to 3 per cent
Recurrence ranges from 2.2 to 13 per cent
Tetany 1.3 per cent; myxedema 4 per cent
Exophthalmos frequently increased
Recurrent nerve paralysis, unilateral 0.9 per cent
Complete cure not available
CONTRAINDICATIONS
Hyperophthalmopathic Graves' disease

TABLE 2

ADVANTAGES AND DISADVANTAGES OF ANTITHYROID COMPOUNDS (CONTINUOUS USE) IN TREATMENT OF HYPERTHYROIDISM

ADVANTAGES
Avoidance of ordeal of operation and surgical risks
DISADVANTAGES
Lasting remission in less than 50 per cent
Reaction incidence 1.8 per cent
Thyroid enlargement not improved
Pressure symptoms and deformities not relieved
Frequent checkups over long periods imperative
Complete cure not available
CONTRAINDICATIONS
Nodular goiter with hyperthyroidism
Large diffuse goiter with hyperthyroidism
Hyperophthalmopathic Graves' disease

had a blood calcium of 7.5 mg. per cent, phosphorus 6.4 mg. per cent.

We have put her on a high nutritive diet, dihydrotachysterol and calcium lactate, and she is free from symptoms. This patient illustrates one of the complications that may occur following surgery but which one does not get from medical management.

THE advantages and disadvantages of surgical treatment and of treatment with antithyroid compounds are listed in Tables 1 and 2. We all know that in every form of therapy there are inherent advantages and disadvantages. The consensus today is that medical treatment can be used in patients who have recurrent hyperthyroidism following surgery; secondly, in the elderly hyperthyroid case who has complications of such severe degree and character, cardiovascular complications, that surgery would not be justified; then again in patients who have mild or moderate hyperthyroidism; and in patients who show no visible thyroid but who have constitutional symptoms, the so-called masked hyperthyroidism that we described some years ago.

We must mention what today is an established practice, and that is the best form of preoperative preparation of the patient is the use of antithyroid drugs and iodine. You can deliver the patient to the surgeon in much better shape and the surgery is much smoother, as is the convalescent course.

Medical treatment should not be used in nodular toxic goiter; it should not be used in large diffuse goiter where the gland is so large that it creates deformity, and in uncooperative patients.

Dr. Robbins will conclude the presentation.

RADIOIODINE IN THE TREATMENT OF THYROTOXICOSIS

ROBERT ROBBINS*

TEMPLE UNIVERSITY SCHOOL OF MEDICINE, PHILADELPHIA

EXTERNAL radiotherapy by roentgen rays and radium has been extensively used in the past for the treatment of thyrotoxicosis, but has fallen, for the most part, into disrepute because of the vicious damage produced upon the structures adjacent to the thyroid. The skin and subcutaneous tissues have been severely and permanently injured by doses of the order necessary to destroy thyroid function. It is possible, however, to deliver such doses to the thyroid by means of internal irradiation with radioactive iodine, and still preserve the integrity of the adjacent structures. Actually, no changes in any

*Director, Radiotherapy Division, Department of Radiology, Temple University School of Medicine, Philadelphia, Pennsylvania.

ROBERT ROBBINS

of these structures have been observed with doses of the order currently used.

The first patients with thyrotoxicosis to be treated with radioiodine were treated by Hertz and Roberts in 1941, and concurrently by Hamilton and co-workers. Many of these patients have been followed in remission for several years, and on the basis of these excellent preliminary results, numerous investigators began to use the radioiodine in clinical investigation. At present, about seventy institutions in the country are engaged in this work, and all have confirmed the good results of these early efforts.

It should be emphasized that the early work was carried out with a cyclotron-produced product, namely I^{130}, with a short half-life, necessitating larger doses than are currently employed. Virtually all modern therapy is done with the pile-produced I^{131}, which has a half-life of eight days, so that the millicurie dosage is considerably less than is indicated in the early literature.

METHOD OF USE

A tracer study of thyroid uptake and retention of I^{131} is performed on all potential candidates for I^{131} therapy, first, because it is a most useful confirmatory diagnostic measure, and second, because our dosage is calculated on the basis of the results of the test study. This entails the oral administration of a small (50 to 100 microcuries) test dose of I^{131}, dissolved in water. Twenty-four hours later a count is made over the thyroid isthmus, using a Geiger-Müller counter. This may be expressed in percentage of the given dose by comparing the count so obtained with the count of a known amount of I^{131} obtained under similar conditions.

Counts are then made over several succeeding days, until the number of counts per minute is less than half of the first day value. From this, a so-called effective (formerly "biologic") half-life may be calculated.

These factors, together with an estimate of the weight of the thyroid gland, form the basis for the calculation of the therapeutic dose. The patient is then given such a dose by mouth in one sitting. The material is tasteless. Appropriate follow-up studies are carried out, with final evaluation of the immediate therapeutic result at six months after treatment.

RESULTS

The results of approximately 500 treated cases are available in the literature, with a probable total of 1,000 cases treated over the country to date. Of these reported and adequately followed, 80 per cent or more have had lasting remissions. A second dose, usually due to low initial dosage, has been required in perhaps 10 per cent of these cases.

Complications are relatively few and transient, consisting of tracheitis, thyroiditis, temporary increase in toxicity, and temporary myxedema. These are minor and often not found except on careful search.

Permanent myxedema has been the most important complication, occurring in 10 to 12 per cent of most series, including our own, but is easily controlled and seems a small price to pay for the good results obtained.

Exophthalmos has been exaggerated after remission in a few instances in most series, its incidence being probably 5 per cent.

HAZARDS

HAZARDS of the use of radioiodine must be considered from the standpoint of the patient, the population, and the physician.

RADIOIODINE IN TREATMENT OF THYROTOXICOSIS

ADVANTAGES	INDICATIONS	DISADVANTAGES	CONTRAINDICATIONS
Remissions in 80 per cent or more	Patient not suitable for surgery	Myxedema in 8 to 10 per cent	Toxic nodular goiter
No mortality	Fear of surgery	Material relatively unavailable	Pregnancy
Insignificant morbidity	Multiple recurrences of toxicity after other forms of treatment	Material hazardous unless properly handled	Pituitary (malignant) exophthalmos (?)
Simplicity for the patient	Injury to vocal cords, parathyroid, from previous surgery	Dosimetry complicated and not stabilized	I^{131} therapy undesirable, but not contraindicated, in (a) children, and (b) patients too ill to await remission from I^{131}
A "leisurely thyroidectomy"	Antithyroid medication contraindicated	Latent period between dose and effect (one to three months)	

From the patient's standpoint, the hazards of future cancer of the thyroid, and of mutations in future generations have been considered. Both these hazards have been greatly exaggerated. A recent survey of the cancer problem by an eminent biophysicist shows that thyroid cancers due to x-ray therapy have in fact not been proved and the likelihood of cancer due to radioiodine is assessed as being insignificant. From the genetic standpoint, the probability of induced mutations by radioiodine is so small as to be unimportant. However, the actual evaluation of these facts must await the passage of time.

Use of radioiodine is fraught with many contamination hazards to the general population and to the physician, and constitutes one reason for the restrictions of use of radioisotopes to qualified research centers.

SUMMARY

Radioactive iodine represents a satisfactory, even an elegant way to produce a lasting remission of thyrotoxicosis in over 80 per cent of its victims, without major sequelae. Its final role in the treatment of thyrotoxicosis, along with surgery and the antithyroid drugs, remains to be evaluated.

FIRST PUBLIC DEMONSTRATION OF ANESTHESIA

ALTHOUGH THERE ARE SEVERAL conflicting claims regarding the discovery of anesthesia, it is generally agreed that the first authenticated demonstration of anesthesia in a surgical operation occurred in the operating theater of the Massachusetts General Hospital on October 16, 1846. At that time, William Thomas Morton, a Boston dentist, administered ether to patient Gilbert Abbott, while surgeon John Collins Warren excised a tumor from beneath Abbott's jaw.

Morton attempted to keep the nature of the anesthetic used a secret, but other investigators had also been experimenting with the use of ether and it was not long before details of the drug and method were generally known.

Thus, within a year, great impetus was given to the practice of surgery by Lister's momentous work on antisepsis and the advent of anesthesia.

An interested observer of the Boston demonstration depicted in Edmund Kopietz' cover painting is Dr. Oliver Wendell Holmes, who is shown on the left facing the patient.

Reprints of the cover may be obtained on request to *Postgraduate Medicine*, 516 Essex Building, Minneapolis 3, Minnesota. Price $1.00.

Diagnostic Clinic

Management of Peptic Ulcer

GEORGE CRILE, JR.*

CLEVELAND CLINIC, CLEVELAND

THE treatment of duodenal ulcer is in a controversial stage at the present time. There is one thing that is perfectly clear, and that is that no matter how successful a surgical operation is, duodenal ulcer is, and will remain, primarily a medical problem. The vast majority of the patients who have duodenal ulcer should be managed medically because the medical results in most cases are excellent.

There remains, however, a certain irreducible minimum of patients who are intractable to medical therapy, and it is important that those patients be managed surgically by the safest and most effective means.

When does a medical problem become a surgical one? That is one of the first and most important issues.

Through the courtesy of Dr. Bowers we have with us a patient who has had ulcer symptoms for fifteen years. The symptoms have been seasonal. The patient has lost up to two months of work a year during the past ten years as a result of the intractable ulcer symptoms. Recently he has had a massive hemorrhage.

To complicate the picture, this patient is stockily built; he has weighed as much as 210 pounds. His weight is down a little bit now. But he is only five feet six inches tall, and operation therefore presents technical difficulties. If you were going to perform a radical gastric resection on this man, you would be confronted with the problem of where to make the incision. Are you going to plan it to give good exposure of the high part of the stomach so that you can do a very radical procedure, giving high protection against the recurrence of ulcer? If so, how are you then at the same time going to expose the duodenum properly so that you can remove and turn in the duodenal stump, which is bound to be a difficult technical job because he has had a penetrating ulcer, penetrating into the pancreas, with hemorrhage?

I think that you can safely say that this problem has become a surgical one, not only from the standpoint of morbidity from time lost from work, but also from the standpoint of mortality, because he is now coming into the age group in which patients with penetrating ulcers and hemorrhage may have fatal hemorrhages. These are the problems which I wish to discuss with you.

We have here also, through the courtesy of Dr. Lemon, another patient, who has a shorter history, but a severe one, upon whom a gastric resection was performed for duodenal ulcer in another city early in 1949. Within six months he developed symptoms of recurrent ulceration. He then had a hemorrhage again. Although x-rays do not in this case demonstrate a marginal ulcer, as they frequently do not, there is no question that from a clinical standpoint this man has persistent ulceration, probably at the stoma.

Here the indications for operation were good, the operation was good, but we have had a recurrence of the ulcer.

I have still another patient I would like to present through the courtesy of Dr. Miller. This lady is a nurse who introduces herself as "Miss Dump Syndrome." She has a history of intracta-

*Cleveland Clinic, Cleveland, Ohio.

Presented November 2, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

ble ulcer pain of fifteen years' duration. She lost three months from her duties as a nurse in 1941, and she has lost three days or more almost annually in these fifteen years as a result of ulcer symptoms. They gradually became more severe. I think when so much time as this is being lost one is justified in having recourse to surgery, although this is a borderline case.

She has had none of the severe complications of ulcer; she was not obstructed; she had not perforated; she had not bled. Yet she was unable, intelligent as she is, under excellent medical care, to render herself comfortable enough to continue work without repeated episodes of loss of work as a result of the ulcer.

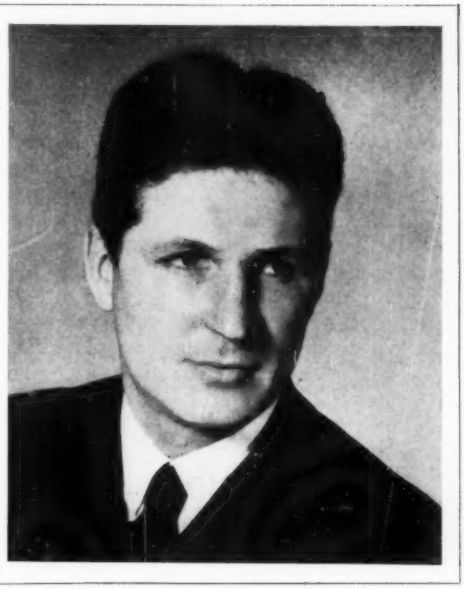
Again I would say that in this type of case the indication for operation is a borderline one which must depend to some degree upon the patient's own desires. We cannot say, "You must have an operation"; we cannot say, "You needn't have an operation." We can discuss it with the patient, point out the economic features involved, and let the patient decide.

A gastric resection was performed and after operation this patient did very well in the hospital, but almost as soon as she returned home she noticed symptoms which she can best describe to you. Would you tell them how you felt after you would eat breakfast?

PATIENT: After I ate breakfast I would become faint and have a desire to lie down. This was usually followed by dizziness, nausea, palpitation, and pressure in my chest. It would become gradually worse and then would gradually become better. I would then have a desire to void and have a dry mouth.

DR CRILE: This is fairly typical of the so-called "dumping syndrome." There may be many factors involved in it. One may be mechanical, overdistention of the jejunum; another may be osmotic; another may be related to carbohydrate metabolism. It is a very real thing following gastric resections or even following gastroenterostomies for duodenal ulcers. This dumping syndrome can completely incapacitate a patient. Fortunately, this patient has made a complete recovery from the dumping syndrome, is entirely well from the gastrointestinal standpoint, with the exception that she is 23 pounds under her previous weight and has not been able to regain her weight, but she has to follow a diet in which she does not drink fluids or take concentrated carbohydrates at her meals.

We must classify her as having a fairly satisfactory result following gastric resection for duodenal ulcer.



GEORGE CRILE, JR.

With this little preliminary I should like to discuss the various methods of attacking the problem of the complicated duodenal ulcer, emphasizing again that the indications for operation are repeated perforations, repeated hemorrhages, obstruction, or intractability where medical treatment cannot keep the patient at his work and he continually loses work as a result of these symptoms.

As you know, the controversy today lies between the advocates of the classic types of operation and those who favor vagotomy. I think it is fair to say that when Dragstedt cut the vagus nerves he started a controversy, and almost everyone almost immediately chose sides. The opponents of vagotomy in general have been the experienced gastric surgeons who over a period of many years have developed a fairly safe and fairly satisfactory technic for gastric resection. These surgeons, often before they had tried vagotomy themselves or had studied the results, went to bat in a critical attack upon the operation, stating that it was unsound, that it

could not work. No one can blame these surgeons, because they had spent a lifetime developing a superior technic for the classical operation, and it was with some reluctance, naturally, that they viewed a simpler operation which apparently could produce as good or better results with a lower morbidity and mortality.

Then there was a group who selected their cases for operation, and I include among these Mr. Maingot, who addressed you last year,* a noted British gastrointestinal surgeon with whom I spent considerable time trying to see where our differences of opinion about vagotomy were. We talked most of a day and finally came to the conclusion that what he had been doing was, in his vast volume of gastric surgery, to select those patients who he said had the "neurogenic" type of ulcer and whom he defined as patients who had very marked symptoms of ulcer but in whom at the time of operation he was able to find very little objective evidence of ulcer. This is very much like the patients who have subjective symptoms of gallbladder disease with pain in the right upper quadrant, but at the time of operation you find no gallstones. We all know that the results of cholecystectomy in that group of cases are very poor. Similarly, if you do not find the expected objective evidence of ulcer and if you select only those cases for vagotomy, naturally you will have very poor results in that selected series.

The other group who have opposed the operation are those who have done only a few vagotomies and who have then perhaps collected a series of cases in their institution or in their town of patients who have been operated upon by a number of different surgeons, perhaps no one surgeon having done more than 5 or 10 of the operations, and I believe that these represent the results of a poor vagotomy. Probably the first 5 or 10 vagotomies the surgeon does will not be as good or as complete as the next 5 or 10 or the first 100 or 200. It would be exactly as if one were to compare the results of gastric resection in the hands of a master with those of some surgeon who is doing his first 5 or 10 operations. So these surgeons have been critical of vagotomy.

The next group who have been critical of it are the students, those like Dr. Moore in Boston, who have done brilliant investigative work on the physiology of the denervated stomach. Here again I wish to point out that this work fundamentally is a physiologic investigation, not a clinical study, because most of these patients have been operated on by the transthoracic approach, which has largely been abandoned because it does not give one an opportunity to evaluate the pathology below the diaphragm, nor does it give one an opportunity to drain the denervated stomach by gastroenterostomy. So these studies, interesting as they are, must be disregarded because the operations were done almost entirely by the transthoracic route.

We have another large group of workers who have made very sincere attempts to study the problem of vagotomy but who unfortunately in their reported tables have not separated gastric ulcer from duodenal ulcer, in spite of the fact that gastric ulcer is an entirely different disease from the standpoint of its etiology and from the standpoint of its response to therapy. Not even Dr. Dragstedt believes that gastric ulcer is a problem which should be dealt with by vagotomy. Almost everyone today agrees that gastric ulcer is best dealt with by gastric resection, except in the occasional extremely high lesion where it would be technically difficult or dangerous to remove it. In these cases some more conservative type of excision with gastroenterostomy possibly coupled with vagotomy is an acceptable compromise, but should be regarded always as a compromise and not as the preferred treatment.

Even Dr. Walters, who made some careful studies of vagotomy, in his reported cases has failed to separate those done by the transthoracic approach, those done for patients who have ulcers which have occurred after a gastroenterostomy with an entero-enterostomy below it (in which cases one would expect ulcer to occur), and those patients who have gastric ulcers. All of these have been thrown into his tables, and yet one can dig out from the text the fact that at least 8 of the 11 cases which he recently reported as failures occurred in the group where you would be very apt to expect the operation to fail.

THE last group of people in this controversy are those who have been the proponents of vagotomy, and these without exception have been the people who have done gastroenterostomies with their vagotomies. It makes no difference if one wishes to consider the operation

*MAINGOT, RODNEY: Surgical treatment of cardiospasm. Postgrad. Med. 5:351-360 (May) 1949.

as being a gastroenterostomy used in the treatment of duodenal ulcer and then protected by the vagotomy to prevent the development of marginal ulcer. This it certainly will do. Vagotomy does protect; just as vagotomy is widely accepted as the treatment of choice for marginal ulcer occurring after gastroenterostomy, so it will protect the gastroenterostomy in the vast majority of cases against the development of stomal ulceration.

It makes no difference, then, whether you consider it as a gastroenterostomy protected by the vagotomy or whether you consider the operation to be a vagotomy in which the denervated stomach is made physiologically functional by draining it so that overdistention of the stomach and retention do not occur. The fact, however, remains that two operations, namely, gastroenterostomy alone and vagotomy alone, neither of which are acceptable in themselves (the incidence of recurrent ulceration is far too high to be acceptable in the gastroenterostomy alone, and the incidence of retention is far too high in vagotomy alone to make it acceptable), when brought together act synergistically to produce an effect which is greater than that of either alone. This combination of gastroenterostomy and vagotomy is preferable to any other form of surgical treatment of duodenal ulcer, both in regard to its ability to cure ulcer and in respect to its low incidence of untoward side effects.

The technic of this operation is relatively simple. Through a midline incision, the stomach is picked up and pulled downward so that the vagus nerves stand out like violin strings. It is not necessary to reflect the left lobe of the liver; it is not necessary to isolate the esophagus. One is not trying to denervate the esophagus; one is trying to denervate the stomach. When one pulls down upon the stomach, the nerves that go to the stomach are not elastic and stand out so that you can palpate them. The blood vessels, the peritoneum, the musculature of the esophagus, all are elastic and do not stand out. You locate the nerves by palpation, pick them up on a nerve hook, strip them up with a little special nerve stripper, up into the mediastinum, resect a segment of the nerve, do the same with the posterior bundle, and carefully search for any additional filaments. Sometimes four or five separate filaments, some only as big as hairs, can be found.

This can be done, as a rule, in about five minutes. One does not need to make a major procedure out of vagotomy if one sticks to the fundamental premises that it is the stomach that one is trying to denervate and that palpation is an easier way to find these nerves than is actual exposure and visualization. Of course, you visualize them too when you cut them, but you find them by palpation.

In regard to postoperative management, that again is simple if a gastroenterostomy is done along with the vagotomy. These patients are ambulatory from the first. The tube is taken out on the first day after operation; it is a nasal tube. The stomach is aspirated on successive nights for two or three days to make sure there is no retention. As soon as we have proved that, no further aspirations are done. Retention diet is started on the second day, a soft diet on the fourth, and in the majority of cases the patient is discharged on the sixth postoperative day, eating a simple diet.

After the sixth week no restrictions are put on these patients at all. They are told that they no longer have an ulcer diathesis, that they can smoke, drink, work as they please, and that they do not require frequent feedings or anything else. They are given the full run of the house, and our confidence in the operation has been well justified by the results.

I SHOULD like to present some figures which indicate the progress of the first 100 patients operated upon and managed in this way. In 100 consecutive patients operated upon, the average duration of symptoms was 12.6 years.

The complications prior to operation in this group of cases were severe and many. Over 55 per cent of the patients had had the severe complications of duodenal ulcer. This group, therefore, does not represent the run of the mill ulcer patient who comes in with a short history and mild symptoms and who is turned over to the surgeon immediately. They represent the patient who has had prolonged trial on medical treatment and has gotten into a great deal of difficulty in spite of that.

Twenty-four patients had pyloric obstruction, 21 had had massive hemorrhages, 9 had had multiple massive hemorrhages, and 10 of them had had perforations.

The patients who had pyloric obstruction had real pyloric obstruction; 20 of these 24 had 30 to 100 per cent gastric retention at the end of six hours. Massive hemorrhage was not considered

massive hemorrhage unless the patient had fainted, had been hospitalized, or had had transfusions. A little tarry stool was not considered a massive hemorrhage. These are the severe complications of ulcer.

Seventy-three of these patients had vagotomy plus gastroenterostomy and the remainder had vagotomy plus pyloroplasty. There is no difference in the results in these two groups of cases.

This group of patients was carefully followed. One patient died in the hospital after operation. One patient died of completely unrelated causes about six months after operation. Three patients were lost track of in the first year and we have not been able to follow them. Of the 95 remaining patients, all have been followed for from one to three years, now an average of two years since operation.

These questions were asked.

"Have you vomited since the operation?" "Yes"—15; "no"—80. Two of these patients vomited repeatedly. One of these is a patient who has a recurrent ulcer which I will tell you about. Three had transitory vomiting just while they were in the hospital, and 10 of the group had vomited only one to six times a year. Vomiting one to six times a year is very little vomiting for patients who have recently undergone a gastric operation.

"Have you had diarrhea?" "Yes"—35; "no"—60. Seven transitory, 20 occasional, 6 mild (less than 4 stools daily), and 2 severe (4 to 6 stools daily). The 20 patients with occasional diarrhea have the same kind of diarrhea that you and I or anybody else is entitled to have occasionally for a few days. None of the patients in this group are incapacitated by diarrhea.

"How much weight have you gained?" Fifty-eight patients gained an average of 20 pounds. We were surprised to find that 8 patients had lost weight, but on further analysis found that 5 of these had been on a reduction diet. The other 3 had lost an average of only 6 pounds since operation.

"Have you had any recurrence of your ulcer-like pain?" A patient who has had ulcer for twelve years knows what ulcer-like pain is. They answered this questionnaire as follows: Three patients had had a recurrence. (One of these has a marginal ulcer.) Ninety-two patients have had no recurrence of their ulcer-like pain.

We have classified the results obtained by these patients as excellent, satisfactory, improved, and failure (Table 1). Excellent results means 100 per cent health from the gastrointestinal standpoint. In answer to a questionnaire or interview, if a patient says he belches after meals or occasionally has a gas pain, that is not 100 per cent health. In spite of these rigid criteria, 62 per cent of these patients have obtained excellent results. A satisfactory result means that the patient has some mild side effects such as the ones I spoke of; he may have an occasional gas pain, a little belching, a little gas or dyspepsia, but he is taking no medicine, following no special diet, and is in no way incapacitated for his work. Excellent or satisfactory results were obtained by 86 per cent of the patients subjected to this operation.

TABLE 1

FOLLOW-UP RESULTS IN 95 CASES OF DUODENAL ULCER TREATED BY VAGOTOMY AND GASTROENTEROSTOMY

	NUMBER OF CASES
Excellent (100 per cent normal health from gastrointestinal standpoint)	62
Satisfactory (no ulcer distress; gastrointestinal symptoms so mild that they do not interfere with work; no special diet or medication used)	20
Improved (no ulcer distress; fewer gastrointestinal symptoms than before operation, but diet and medication, or both, have been used to control symptoms)	10
Failure (persistence of ulcer-like distress or development of other gastrointestinal symptoms sufficiently severe to constitute disability)	3
Excellent plus satisfactory	86 per cent

TEN of the patients are improved. By improved we mean they have no ulcer pain but they have gastrointestinal symptoms of sufficient severity to require some kind of diet or medical management, or at least to make the patient believe that a special diet or medical management is desirable. The symptoms may be referable to irritable colon or associated intra-abdominal disease, but in any case although the patients are not 100 per cent well, and are following some form of medical treatment, they do not have ulcer symptoms.

Ninety-seven per cent of the patients have no ulcer symptoms after this operation, are working, and are well.

If the side effects are sufficiently severe to be incapacitating, we classify the result as a failure, and we classify any patient who had recurrence of his ulcer pain as a failure. Three per cent of the patients in this group have been so classified.

We have compared this group, applying the same criteria, to a group of gastric resections. In this group of gastric resections, 77 per cent were considered to be in the excellent and satisfactory group as compared with 86 per cent in the group subjected to gastroenterostomy and vagotomy. Four per cent were considered improved, and there were 19 per cent whom we had to consider failures because of either severe side effects such as the dumping syndrome or recurrent ulcerations such as occurred in the patient I presented. In 3 of these 87 patients whom we followed after gastric resection in the same way that we followed the first group of patients, marginal ulceration occurred within the first eighteen months after operation.

We have now performed vagotomy and gastroenterostomy in over 400 consecutive cases of duodenal ulcer. In this group of patients the mortality rate has been extremely low. We believe that it is going to be about 0.5 per cent. We had 1 or 2 fatalities earlier in our experience when we were developing our technic, but we have now operated upon 259 consecutive patients without a death, performing vagotomy and gastroenterostomy or pyloroplasty. This is the longest series of operations for duodenal ulcer which we have ever been able to perform at the Cleveland Clinic without a fatality.

In conclusion, I think I should urge that before abandoning vagotomy one should consider that gastric resection entails a higher mortality than does vagotomy and gastroenterostomy; the mortality rate of gastric resection runs between 2 and 3 per cent in the hands of competent surgeons, and I believe that the mortality rate of gastroenterostomy and vagotomy is certainly going to be less than 1 per cent in similar hands.

The difference between these two operations is the danger of dealing with the duodenal stump. That is the great disadvantage of gastric resection, that is where the majority of severe complications occur, and that is the cause of most of the deaths. So I think before abandoning vagotomy and gastroenterostomy, particularly in such a case as the first patient that I presented, the rather obese man who has severe ulcer symptoms, one should consider the mortality, the morbidity, and the side effects, which in our experience have been lower following vagotomy and gastroenterostomy than following gastric resection.

Finally, the curability rate of the ulcer and of the ulcer symptoms has been higher following vagotomy and gastroenterostomy than following gastric resection. Before we abandon vagotomy I think we should make perfectly sure that we have given vagotomy and gastroenterostomy a fair trial, first of all making sure that the surgeon performs either a pyloroplasty or a gastroenterostomy along with the vagotomy because the results will not be satisfactory if you don't do that; secondly, make sure that you have tried it in a consecutive series of cases and have not selected a group of patients who have the minimum objective evidences of ulcer and who may perhaps not have had ulcer at all but may have had some functional gastrointestinal disease. Do it with a gastroenterostomy, do it in a consecutive series of cases and, finally, make sure that the surgeon who is doing this operation has had sufficient experience with the operation to have learned the little tricks of the trade which make vagotomy complete and effective.

SUMMARY

1. Vagotomy alone is not a satisfactory treatment for duodenal ulcer.

2. Gastroenterostomy alone is not a satisfactory treatment for duodenal ulcer.

3. Vagotomy and gastroenterostomy act synergistically and in our experience this combination is the safest and most effective surgical treatment of duodenal ulcer.

4. The results in 100 consecutive cases of duodenal ulcer treated by vagotomy and gastroenterostomy are reported. Ninety-five of these have been followed for twelve to twenty-nine months and the results have been classified in Table 1.

Diagnostic Clinic

Carcinoma of the Larynx

LOUIS H. CLERF*

JEFFERSON MEDICAL COLLEGE, PHILADELPHIA

It may appear presumptuous to interest an audience made up largely of general practitioners in a single clinical entity involving such a small organ as the larynx. On the basis of an experience gained in studying the records and examining the larynges of over 1,700 cases of carcinoma of the larynx observed in the Department of Laryngology and Broncho-Esophagology at the Jefferson Hospital I have observed that a majority of these patients at some time or other have consulted their family physicians. In many instances the patients were referred to a laryngologist for diagnosis and treatment; in a number, however, no examination of the larynx was done, symptomatic treatment was carried out, and a diagnosis was made late.

It would seem important, therefore, that every physician who treats patients complaining of hoarseness or other symptoms referable to the larynx should be familiar with mirror laryngoscopy and should be aware of the possibilities and refer the patient to a laryngologist. Obviously it is impossible to know the entire field of medicine but one should know much about many things to advise patients intelligently.

Of this large group of 1,700 cases, slightly more than 50 per cent were treated surgically. The remainder either were treated by irradiation or were considered as hopeless cases and received palliative treatment. Since a large number of these patients were seen when the disease was practically hopeless it would appear advantageous to discuss a few of the diagnostic problems so that a diagnosis can be made early.

While the larynx is commonly considered as being difficult to examine it is nevertheless readily accessible for study by mirror laryngoscopy or direct examination. Further, the results of treatment in early carcinoma of the larynx are better than those secured in any organ of the body, the skin excepted. What may be said about the problem of carcinoma of the larynx as it affects the general medical man? The most important problem is to recognize carcinoma. It cannot be diagnosed by inferential means. One therefore should be familiar with symptoms as well as the methods of diagnosis.

Since in approximately two-thirds of the cases of carcinoma of the larynx the growth involves a vocal cord the earliest symptom is disturbances in the voice. This is particularly important in the case of men over 40 years of age since practically 90 per cent of all laryngeal carcinoma occurs in the male. A good working rule would be to suspect carcinoma in any man who has been hoarse for more than four to six weeks. While not every one would have carcinoma it nevertheless would suggest to both patient and physician that the larynx should be examined. There are many benign lesions, such as vocal nodules, polyps, papilloma, contact ulcer, as well as such systemic conditions as tuberculosis and syphilis which involve the larynx, but a diagnosis of all these can be made promptly if appropriate measures are instituted.

*Professor of Laryngology and Broncho-Esophagology, The Jefferson Medical College of Philadelphia, Philadelphia, Pennsylvania.

Presented November 2, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

LOUIS H. CLERF

Carcinoma is a painless disease in its early stages. When pain occurs it more often is referred to other structures, notably the ear, and is not limited to the larynx. Dyspnea or shortness of breath results from obstruction to the airway. This always is indicative of an advanced lesion.

It always is disturbing to find a patient with carcinoma of the larynx of many months' duration who has been treated as a case of chronic laryngitis or chronic hoarseness. While chronic laryngitis is a distinct entity it should not be considered diagnostically until other lesions have been ruled out. Chronic hoarseness simply means that the patient has had hoarseness for a long time. This is not a clinical entity but is merely a symptom.

I would like to discuss some examples of laryngeal lesions of epithelial origin some of which are not malignant but must be distinguished from carcinoma.

In one case there was a lesion which appeared papillary and was not ulcerated. It occurred on the right vocal cord of a man 44 years of age. The gross appearances suggested papilloma but one must always beware of papilloma in adults. A biopsy was performed and a diagnosis of papilloma was corroborated. Papilloma may appear as single or multiple lesions. They frequently recur after removal, particularly in children. In adults they may occasionally undergo malignant changes.

In the next case there was observed a whitish, irregular-appearing lesion on the edge and upper surface of the vocal cord of a man, aged 49 years. The history here is important for this man had been an inveterate smoker. While one cannot state definitely that excessive smoking is a predisposing factor in keratosis or carcinoma, it has been our experience that a majority of the cases of keratosis of the larynx are inveterate smokers. This proved to be a case of keratosis.

The essential difference histologically between keratosis and carcinoma lies in the behavior of the cells along the basement membrane. There is proliferation of cells in both cases but in keratosis there is exfoliation and the cells do not penetrate the basement membrane. Keratosis, however, should always be considered as potentially malignant for many cases do ultimately develop malignancy as is shown in the next case, a man 54 years of age, an excessive smoker who had keratosis of both vocal cords. On the left cord anteriorly there was a slight change in the character of the lesion, however, and on biopsy this proved to be carcinoma. This was an early lesion and the case was treated successfully by dividing the thyroid cartilage in the midline anteriorly and removing the carcinomatous vocal cord.

Another case was that of a classical early carcinoma involving the vocal cord of a man. The lesion was a nonulcerating nodule involving the edge and upper surface of the cord at the junction of its middle and anterior thirds. Strangely this is a common site for carcinoma of a vocal cord but it also is the customary site for vocal nodules and polyps. This patient had been hoarse about nine weeks. The prognosis in this type of case if treated promptly is excellent; in fact one should have at least a 95 per cent nonrecurrence rate after five years.

We now consider advanced cases of carcinoma involving the entire vocal cord from the anterior to the posterior commissure. In the first case there is encroachment on the glottic chink producing some shortness of breath and there is fixation of the arytenoid. The patient has pain but this is referred to the ear al-

FIGURE 1. Schematic drawing of view of larynx secured by mirror laryngoscopy. There is an ulcerated nodule involving the edge and upper surface of the left vocal cord in its anterior and middle thirds. Biopsy revealed carcinoma. This is an early lesion and is ideal for laryngofissure. (See Figure 2.)

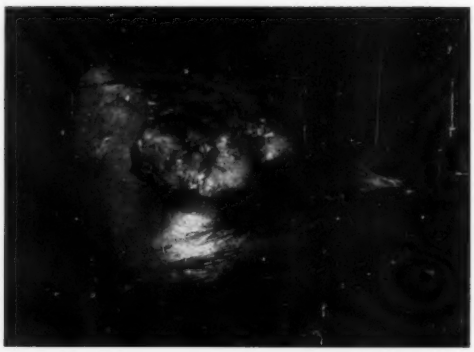


FIGURE 2. Specimen removed at laryngofissure consists of the vocal cord with a small superficially ulcerated nodule involving the edge and upper surface. The nodule is surrounded by a wide margin of normal tissue insuring complete removal of the primary lesion.

though the ear itself is normal. With a history of hoarseness of eleven months' duration the diagnosis should have been made many months ago. It is now an advanced lesion and one must remove the entire larynx if surgical treatment is to be carried out. In addition the lymph nodes along the side of the neck should be carefully investigated for evidences of metastasis since carcinoma of the larynx commonly metastasizes to the cervical lymph nodes of the involved side.

The next case is one of carcinoma involving the epiglottis. This patient complained of a sticking sensation in his throat and at one time believed that he had swallowed a fish bone. There were no voice changes until recently for the growth did not involve the vocal cords. The recent hoarseness has resulted from encroachment of the growth on the cords interfering with their movement and vibration during phonation. This patient too will require radical treatment.

To summarize briefly thus far, it should be very apparent that any lesion on a vocal cord interfering with approximation and vibration of a cord will produce hoarseness, a symptom that can be readily elicited. This should call for investigation by mirror laryngoscopy. Carcinoma involving other portions of the larynx does not produce hoarseness as the initial symptom but these too require examination and no study is complete until the larynx has been inspected.

Three patients whom I have treated surgically have graciously agreed to appear before you.

Case 1 is a man, 55 years of age, who had been hoarse for three months before his larynx was examined. There was found an ulcerated nodule on the edge and upper surface of the vocal cord anteriorly as shown on the schematic drawing (Figure 1). Biopsy was done and the diagnosis of carcinoma was corroborated. A laryngofissure was performed by splitting the thyroid cartilage in the midline and removing the involved vocal cord (Figure 2). The patient now in his ninth postoperative year has continued his work in the storage battery business, feels well, has a good voice, and now will address you. (Patient talked to audience over microphone.)

The next patient, a barber, now 68 years of age, became hoarse in December, 1943, and was not diagnosed as having carcinoma until one year later. The lesion was extensive, producing marked hoarseness and some shortness of breath. It was necessary to remove the entire larynx, which is shown in Figure 3. He learned to talk by swallowing air and belching it. As a result he has been able to continue with his trade as a barber and to talk to his clients as he now will talk to you. (Patient talked using pharyngeal voice.)

The third patient, also an extensive case of carcinoma, had a hoarse voice only for nine weeks, although he had symptoms of sore throat for one year. The carcinoma involved the epiglottis and did not interfere with phonation until it extended down to and rested on the vocal cords, interfering with their approxima-

FIGURE 3. Specimen of larynx removed at operation reveals an extensive carcinoma involving the entire left vocal cord with extension subglottically. There was fixation of the vocal cord as well as arytenoid. This lesion, starting on a vocal cord, was at one time small and could have been treated by a more conservative form of surgery.

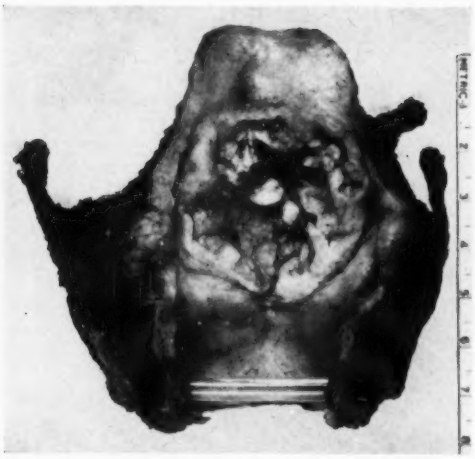


FIGURE 4. Larynx removed at operation reveals a large ulcerated, fungating carcinoma involving the entire laryngeal surface of the epiglottis. The growth has invaded the pre-epiglottic space. Voice disturbances were recent as the growth does not involve the vocal cords but acts as a damper.

tion and vibration. It was necessary to do an extensive removal of the larynx together with the hyoid bone and the pre-epiglottic space (Figure 4). This patient has developed an excellent voice and has been very helpful in teaching others to talk. (The patient talked to the audience stating he was operated on June 6, 1946. He began to talk fifteen days after operation and seven weeks after operation went back to his work as a lineman. He still climbs poles, works on steel towers and does all of the work he ever did. He was happy to help others to talk and visits the Bronchoscopic Clinic once or twice every week to help.)

I HAVE presented 3 patients with carcinoma who have exhibited three different lesions and two plans of therapy. In the first case, an early vocal cord carcinoma, it was possible to do a laryngofissure. The patient has a normal airway and a good voice. The prognosis is excellent. This surgical procedure can be used only in cases of early carcinoma. In the last two cases the carcinoma was advanced although the sites were not the same. Complete removal of the larynx was required in each case. While the airway is adequate, it opens on the front of the neck. Both patients have good voices although obviously they are not normal. Both, however, are free from carcinoma. In the third group where the growth no longer is confined to the larynx but has metastasized to the cervical lymph nodes the situation is more serious. We now do a laryngectomy and block dissection at the same operation. A number of these patients have passed the five-year period without recurrence.

Obviously no discussion of the therapy of cancer would be complete without referring to irradiation. Any of these lesions can be treated by this method. As with surgeons so also with radiologists the results of treatment vary. In this clinic surgical treatment is favored even when radiologists are consulted, for the results by surgical measures have been more satisfactory. It must be recalled that we cannot try one method of treatment and if it is unsuccessful, try another and assume that the same end results can be secured. The plan of treatment that promises the greatest number of living patients without recurrence after five or more years is the one that should be favored.

Diagnostic Clinic

Rehabilitation of Common Neuromuscular Disorders

GEORGE MORRIS PIERSOL*

UNIVERSITY OF PENNSYLVANIA GRADUATE SCHOOL OF MEDICINE, PHILADELPHIA

THE purpose of this clinic is to demonstrate what a carefully integrated and patiently carried out rehabilitation program can do for individuals who by reason of injury or disease have been so badly crippled that without further aid they would become complete charges on the community and individuals with discouraging futures.

The lessons learned from the Army's great rehabilitation program, with which many of you are familiar, in which many of you actually took part and which has been carried on by the Veterans' Administration, have now been applied to the problems of civilian practice.

There was a time when individuals who suffered serious injuries or diseases which left them with disabling residual changes in their nervous systems and muscular apparatus were practically doomed to a hopeless existence. Thanks to the example set by Dr. Howard Rusk, Dr. George Deaver, and their associates, it is now apparent that if a suitable systematic program is adopted by civilian institutions such patients can be returned to various degrees of usefulness, as was so well demonstrated in our armed forces. Our purpose is to show you several individuals whom we have had under our care for considerable periods of time—patients who when we received them were either bedridden or doomed to wheelchair lives, and were unable to care for themselves.

These 4 patients have one disability in common. They had complete loss of power in their lower extremities. As a result, they were unable to stand and unable to get themselves about except in several instances by the use of a wheelchair.

The first problem in the rehabilitation of these individuals was to get them to navigate, so they can move around. A person who is down, unable to stand, balance, or get from place to place is under a tremendous disadvantage. The patients we are about to show you present that difficulty, which has been our main concern.

The attention of those who care for these people cannot be directed merely toward their lower extremities, or any single part that is involved; the *whole* patient has to be considered. Efforts have to be made to build up their remaining good musculature to the point where it is able to carry a much greater load than normally it would be required to do.

The first patient is a young woman who suffered from poliomyelitis in September, 1945. She was in the Philadelphia Hospital for Contagious Diseases for a long time. The first twenty days of her stay there were spent in a respirator because of involvement of her accessory muscles of respiration and abdominal muscles.

She was given the conventional hot pack treatment and muscle retraining for a period of about four months and was sent to her home in August, 1946. At that time she suffered a complete flaccid paralysis of both lower extremities. She also had marked weakness of the

*Professor of Medicine, University of Pennsylvania Graduate School of Medicine; Professor of Physical Medicine, School of Medicine, and Director of the Center for Research and Instruction in Physical Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.

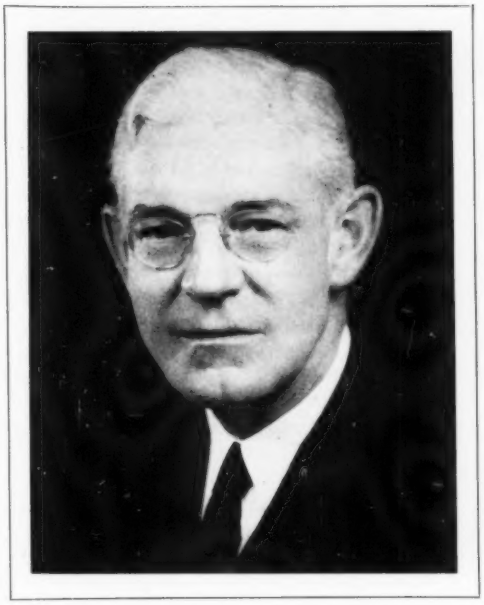
Presented November 3, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

right side of her shoulder girdle involving the right deltoid, and in addition to that almost complete loss of power in her abdominal muscles. Thanks to the generosity of the local chapter of the National Foundation for Infantile Paralysis, she was given physical therapy by competent physical therapists at her home from April, 1948, until September of that year.

At that time she was transferred to the Institute for Rehabilitation in New York, where she came under the supervision of Dr. Deaver and his efficient group. They evaluated her and concluded that this girl's first problem was to acquire the ability to stand and ambulate. She was given long leg braces with a pelvic band, and got around entirely in a wheelchair. Muscle tests showed that she had flail hips, flail knees, and zero muscular activity in her trunk muscles, with a marked weakness of her right shoulder girdle and her right elbow. She was able to perform tasks of daily living; by which is meant doing things such as eating, combing her hair, and going through the ordinary program of making a toilet with a considerable degree of ability. In that respect she had a fairly good outlook so far as her self-care was concerned. She did not have ability to elevate herself, to get herself up out of a chair, or to stand. That was the condition which she presented when she first came under our care.

After six months in New York, she was sent back to the orthopedic department of the University Hospital, and they in turn referred her to us to initiate her rehabilitation program. The problem was to strengthen her abdominal and trunk muscles, the flexors and extensors of her arm and shoulder girdle, and then develop in her the ability to get out of a chair and ambulate. This has involved a program extending over a great many months. During this time she has had assistive, passive, and resistive exercises to her lower extremity as well as manipulations designed to stretch her heel cords and her hip flexors. Subsequently, after this had been accomplished to some extent, she was given thorough training in crutch walking and further training in the activities of daily living. Muscle checks done recently show that some of the muscles which previously were zero now show a slight degree of return of power, but very little.

This case illustrates that in spite of the conventional idea that after a certain number of months individuals cannot regain power, progress can be made. We have demonstrated repeatedly that even years after the initial injury, a well carried out program may bring about return of power in muscles which were thought to be entirely in a zero state.



GEORGE MORRIS PIERSOL

I shall ask the patient to demonstrate now just what she can do. If you observe this patient getting out of the wheelchair, you must be impressed by the tremendous amount of energy she expends in so doing.

It has been shown that individuals with appliances, in making ordinary effort such as getting out of a wheelchair, will expend energy many hundreds of times greater than that which a normal person requires to perform the same act. She is a good example. You will also notice as she stands that she has a very marked lordosis. One may say, "Her treatment cannot have been very successful because of the deformity in her back." At one time she wore a night spinal brace. This has been abandoned recently at the suggestion of the orthopedists, and at the patient's request because she has found that this lordosis gives her no discomfort. With a brace she is inhibited and unable to do many things that she can do without it, although theoretical-

ly it would seem as though she should be wearing a spinal brace. From a practical standpoint she does much better if it is omitted, which brings out another lesson: that you cannot be governed too much by conventional and traditional practices.

Our primary purpose is to get people back to a point where they can do the maximum amount and take as much care of themselves as possible. In order to do that, as Dr. Deaver has repeatedly emphasized, one must be prepared to abandon preconceived notions and be governed chiefly by practical considerations.

Here is a girl who several years ago was doomed completely to a wheelchair existence. She has demonstrated now that she can get out of her chair with braces and crutches. She has also demonstrated the efficient way in which she has learned to crutch walk.

Another important problem is to teach these people to get upstairs and to go up even a small elevation such as a curb. The practical aspects of this are shown in this patient. At one time she had the opportunity to get a position. She was unable to take it because where she was to work it was necessary for her to get up one step. At that time it was impossible for her to do that. Now she has been working on that problem. The height of this platform is the height of a low curb. She will show how she can get herself up over a curb, turn around, square away, and proceed under her own power. Again, you will note the tremendous amount of energy output required to carry out these simple procedures.

She has now advanced to the point where if she is in a house where there is a step or a high sill to get over, by backing up to it and by properly using her crutches and her shoulder muscles, she is able to get herself up.

When she wants to move rapidly she has learned a swing-through gait, as it is called, with her crutches. When she is not in a hurry she has acquired what is known as a four-point crutch gait. Although she accomplishes these acts now with great dexterity, it must be remembered that it required a tremendous amount of patience, determination, and energy on her part, to learn to do these things. She has reached the point where so far as physical rehabilitation is concerned we have helped her about as much as we can.

The next step is her vocational rehabilitation. She is extremely well educated and an intelligent young woman. The State Rehabilitation Group is now investigating ways and means by which she can be trained to do typing and bookkeeping. She already assists her father, who has a good retail distributing business, in those activities. In spite of the fact that a few years ago she had and still has complete loss of power in her lower extremities she has now reached the point where she takes care of herself at home, is not a charge on her family, and is actively engaged in a productive occupation, which, after all, is the ultimate objective of individual rehabilitation.

This boy is another victim of poliomyelitis. In 1945 he had complete involvement of his lower extremities, his back, his abdominal muscles, and the anterior part of his deltoids. He also received physical therapy at home for a number of years. When he came to us he was brought on a litter. He was unable to move his lower extremities. He had spent practically the previous two years in bed and had, in addition to the disability incident to his disease, that due to atrophy which comes from too long bed rest. You have all heard in the last few years of the evils of prolonged bed rest. When we saw him he showed the atrophies of disuse throughout his entire musculature. He had a marked scoliosis with extreme rotation of his thoracic and lumbar spine. Both his hamstrings and his heel cords were markedly shortened with pronounced flexion contracture at the hip and the knees. He was absolutely incapacitated.

He was sent to us with directions to get him to walk. That was the main objective of his family and of the orthopedist who had him in charge. The first step with this individual, before we could get him up or teach him to walk, was to develop properly his shoulder girdle and arm muscles.

I well remember after he had been under treatment for some months that his family was somewhat disgusted with us, and even the orthopedists were not very sympathetic because they said, "We sent this boy to you to teach him to walk. He has been with you all this time and he hasn't learned to walk yet." At that time he was receiving therapeutic exercises, particularly to his upper extremities and stretching for the contractures of his hip and knees. In this boy, as in similar cases, before he can be erect or use crutches you must develop

the musculature involved in such activities.

Finally, after his shoulder girdle was in shape so that he could be gotten up and on crutches, he was equipped with long leg braces with a pelvic band, and was taught to stand and to balance—one of the most important things. Then he was taught crutch walking, and later he was trained in the various activities of daily living.

Here he is, another case of poliomyelitis with loss of power in the lower extremities. He has adapted himself and shows certain differences in contrast to the first case. He has a different crutch gait which he has been able to develop in which he uses what slight return of power there has been in his lower extremities and his arms. He can do a great many things, and I am going to ask him now to demonstrate some of the activities that he has learned.

First is his ability to walk. He has a modified three-point crutch gait. This he has worked out himself in order to utilize all the muscles that he has. His method of getting up over a curb is different from the first case. He gets out of the wheelchair with a tremendous effort but perhaps more easily than the other patient. You would think that he had some quadriceps left, but as a matter of fact he has no quadriceps at all.

This boy has advanced to the point where instead of being a helpless invalid lying on a stretcher, we actually have been able to employ him as a technician in the laboratory. He can get around perfectly well on his crutches. For any long hauls he still uses his wheelchair. He comes to the hospital in a taxicab, gets himself out and to the laboratory. There in his wheelchair, having a mechanical bent and good use of his hands and forearms, as well as being extremely intelligent and ambitious, he does a very good job as a technician. He, too, is about to start on a course of vocational rehabilitation, sponsored by the State Rehabilitation Board, in which his mechanical genius will be put to good use. He, too, has now reached the point where he can look forward to a reasonably self-sustaining existence, not the hopeless vegetative life of the cripple that he was when he first came under our observation.

The next 2 patients are young men who have not had poliomyelitis but in the course of their civilian activities, have suffered accidents which have brought about serious transverse lesions of their spinal cords.

This boy suffered a gunshot wound in his right chest in March, 1947. This brought about a transverse myelitis at the level of the tenth and eleventh thoracic vertebrae. As a result of the bullet wound he was completely paralyzed from the waist down. On examination it was found that the bullet had entered his chest at about the sixth rib in the anterior axillary line and lodged just to the right of the spine. Laminectomy was performed under local anesthesia between the eighth and the eleventh thoracic vertebrae and a bone chip was found pressing on the cord at the level of the tenth thoracic. This was removed and the cord was found, as one might expect, to be markedly contused, very much swollen, but apparently otherwise intact. Nevertheless, definite organic damage had taken place.

About eighteen days after he was admitted for operation he had the misfortune to develop massive phlebothrombosis of both lower extremities. Following this, plus the nutritional disturbances incident to the cord lesion, he developed extensive decubitus which involved his thighs and extended down over the sacrum. This has been a tremendous factor in retarding his rehabilitation program. For a long time, all that could be done with him was to try to clear up this decubitus. He had repeated operations, including skin grafts, over his sacral area. Even now we must watch him very closely because if any undue pressure is exerted it causes breaking down of superficial tissues and a fresh ulcer.

He reported to us in June, 1948, with complete sensory loss below the level of the first lumbar on both sides and absence of all motor power in both lower extremities. He had, in addition, marked flexion contraction of both his hips to the extent of about 35°, some ankylosis of the right knee, with only about 20 per cent of motion in the left. This was due to shortening of his hamstrings and heel cords, in addition to marked contractures which developed in the fascia and subcutaneous tissues as the result of the extensive decubitus and the scars that had resulted from their healing. We were then faced with the necessity of trying to stretch these contracted limbs before we attempted anything else. Passive stretching was undertaken. He was put in a Hubbard tank where he had the advantages of heat and the mechanical effects of water. Gradually it was possible by resistive exercises to his upper extremities to pre-

pare him so that he could start ambulation and develop increased power in the muscles of his arms and shoulders. In the meantime, we overcame the contractions to a considerable degree.

Because of the irritation due to the cord lesion he also has from time to time involuntary mass movements of both lower extremities with severe contractions which almost double him up. For that reason, off and on we have had considerable difficulty with him. He has been fitted with long leg braces with a pelvic band and has learned very successfully to balance. After a course of gait training he is now able to get around, to carry out at least the activities of daily living, and is not immobilized in a chair or on a litter.

He has learned well the ability to balance. He walks with a three-point gait, and we will have him demonstrate the way he can balance on his crutches and walk. He will show you how he has learned, with his crutches either forward or backward, to balance. It is quite a trick which some patients acquire very well. In addition, he has devised a three-point type of crutch gait.

His legs are still unable to flex very much. When he is in a wheelchair they have to be kept fairly straight. This adds to the difficulty of getting around even in an automobile, but he manages. He can swing himself laterally. That does not seem to an able-bodied individual as though it amounted to very much. If you had been in the predicament where you could not even stand, let alone get around, you would know that it is a great accomplishment. He is able, by putting his crutches back, to balance just as well as forward. So he has really acquired that art very well.

THE final patient had an automobile accident in February, 1947, in which he suffered a transverse myelitis at the level of the twelfth thoracic and first lumbar, also a fracture of his right femur, and was hospitalized until August, 1947. He was sent home where he received minimal treatment. On admission to the University Hospital in June, 1949, he had, as the preceding case had, a cord bladder which required the care of a urologist, as well as complete flaccid paralysis of the entire right leg and left lower leg, bilateral footdrop, and atrophy of the musculature of his legs.

He has been equipped with a long leg brace on the right leg and a short one on the left leg. He was started on a very intensive program which began with general reconditioning mat exercises, followed by gait training and the activities of daily living. As a result he has acquired a great deal of ability to get himself around. He probably can get along faster than any of the patients under our care. In addition, he has learned several things which are very important. He has acquired the art of falling, and having fallen, how to get up. Among other things, these patients must be taught how to fall without hurting themselves. If they fall and hurt themselves a psychic block develops. Then it is hard to get them to attempt anything. If, however, they have acquired confidence and know that if they do go down they are not going to hurt themselves and can get up again, it gives them a tremendous feeling of security.

Jim, how about showing us what you can do? He has several ways by which he can get down. The method he just used is the most abrupt and most rapid way. Note the fact that as soon as he starts to fall he does an important thing—he throws his crutches away from him. Patients are all taught to do that, so that their crutches are no longer in the road; they cannot fall on them and do themselves any further damage. He is able to get himself up without any trouble.

How else can you get down? They learn when to lock and unlock their braces at the knee or hip, as the case may be.

He also has acquired a very good swing-through gait.

You see, there is more than one way to "skin a cat." If they can't learn to get down one way, you can teach them to get down another. He has been particularly dextrous, very energetic, and is quite the past master at getting down and getting himself up. The answer is, of course, that he has good strong shoulder girdle and arm muscles and has learned how to get the maximum good out of them. He also has mastered getting over curbs. He does steps and curbs really well. He can go along quite rapidly with his swing-through gait, swing up on a curb, and get down again. He feels independent now about many things, justly so. In spite of his disability he has reached the point where he is an extremely useful citizen and no longer a drag upon his family.

Scientific Exhibit

Diseases of the Nails

CLEVELAND J. WHITE

PROFESSOR AND CHAIRMAN, DEPARTMENT OF DERMATOLOGY, LOYOLA UNIVERSITY COLLEGE OF MEDICINE, CHICAGO

Nails can be involved by both local and constitutional factors, as well as congenital malformations. The effects of general diseases and the trophoneuroses are given under the domain of general medicine and the details can be found in any textbook of that type. Congenital malformations are manifested in different ways, as by complete absence of nails, or by replacement of nails by horny masses, as in the atopic, hypertrophic, and clawlike deformities. These congenital malformations are easily recognized and are well known. Dermatologic aspects of nail deformities come largely under the heading of local causes as various traumatisms, infectious diseases, and localizations of various dermatoses.

RINGWORM (ONYCHOMYCOSIS)

Ringworm or infectious vegetable parasitic diseases of nails have become very prevalent and important. There is local nail destruction and a place is formed for harboring fungi which reinfect the glabrous skin, thus causing an ever-increasing number of ringworm or so-called "athlete's foot" infections.

Ringworm of the nails is caused roughly by three types of organisms: (1) the hypomycetic group as the Trichophytons, (2) the yeast-like group as the Monilia organism, and (3) in rare instances, by the favus organism. Clinical characteristics of ringworm of the nails vary from moderate scaling in the lateral folds of the nail to complete nail destruction. Usually it begins as a scaling of the free extremity of the nail plate or one of the folds.

The infection may start either with or without any cutaneous manifestation. One nail is usually involved first and then there is symmetrical progression until any number may be involved. The infected areas become heaped up and assume an opaque appearance. The distal portions become powdery and fungi can be found early under the microscope after digestion of some of the debris by potassium hydrate in 10 per cent strength. Severe cases show almost complete nail destruction. An occasional case will not show any gross nail-plate disintegration, but will become separated from the nail bed.

With the exception of the monilial type of infection there is practically no pain involved in ringworm of the nails. The distinguishing clinical finding between monilia and the ordinary ringworm infection is that the former is usually accompanied by a nonsuppurative paronychia. The latter type is typical of the infected fingers in fruit packers and handlers.

Because of the close resemblance of other nail syndromes, especially psoriasis, it is well to confirm every suspected case of ringworm by microscopic examination. As the cultural findings are only positive in roughly 20 per cent of the cases, it can be easily seen that they are only of academic interest. A diagnosis then is made by the clinical findings supported by laboratory studies. The biologic tests are of practically no value in making the diagnosis of fungous diseases of the nails.

As for treatment, the most valuable preparation is Whitfield's ointment used in double strength and applied after mechanical scraping. The x-ray in fractional doses is very valuable in ordinary ringworm type and the grenz ray is of value in the monilial and yeast-like types. Surgical removal of the nails, in my opinion, is never indicated for fungous infections because the infection almost invariably appears in their regrowth after removal. At times other ingredients are used in ointments in various combinations.

PSORIASIS

Psoriasis of the nails can occur independently of glabrous skin involvement. The five principal changes observed in psoriasis of the nails are: (1) detachment of the nail; (2) partial parenchymatous alterations likely to end in partial destruction of the nail; (3) color changes; (4) shortening of the nail, and (5) nail puncta.

In nail detachment the free edge loosens first at about the length of 2 to 7 mm., more often from

NOTE OF APPRECIATION. *Miss Charlotte S. Holt supervised the preparation of the medical illustrations. Assisted by Miss Alice H. Nelson, she arranged the artistic format which enabled the original exhibit to win first prize in the Scientific Exhibit of the American Academy of Dermatology. Many dermatologists have forwarded resumés of instructive nail cases with photographs and full credit is given. Dr. Robert H. Harris took some of the photographs.*

3 to 4 mm. The nail is elevated in the process, leaving a short space between the nail and its cutaneous bed large enough to collect dust. The nail substance thins down and becomes a yellowish-brown. Rarely is keratotic debris seen adherent to the nail bed, and the notable hyperkeratosis of syphilis (paronychia) is never seen. The nail extremity seldom passes beyond the digital pulp, the nail being shortened. It also has less "urge" to grow.

The color, usually yellowish or brown, seldom affects the nail. It occurs either on the free edge of the detaching nail or at the side in a small, circular concavity or pit facing the nail exterior. To this color change a friability of the nail is sometimes added. This is evidenced by elevation and only occurs when discoloration is already present. Finally, the nail ulcerates, either along its sides or toward the lunula, or "half moon," at the base. If it ulcerates along the free edge, it shrinks down to a mere 2 to 4 mm. in length. The edge of this shrunken nail is yellow, it is elevated from its bed, and the nail bed itself shows a little unimportant keratotic debris.

The punctate erosions, which are not so common as the conditions just described, consist of small, thimble-like depressions analogous in fineness to the punctate erosions encountered in eczema. They are almost always extremely numerous when they do occur, so they can be readily distinguished from the nail erosions of syphilis, where they are least numerous. The existence of these psoriatic erosions implies that psoriasis is a disease of internal origin and that the causal agent begins to multiply at the nail matrix, led there when the psoriasis passes into the erythrodermic phase and attacks the fingers.

The nails are not slow in presenting a transverse line, which indicates the initial onset of generalized psoriasis. As the nail continues to grow, it is progressively eliminated, leaving in its place a shortened nail, altered in structure by the erythrodermia of the nail matrix. Here, too, as in all erythrodermias which are infectious or toxic, the striae appear at the same height and same distance from the nail matrix. In the final stage the nail is shortened, softened, and somewhat atrophic in structure. The nail of normal consistency in being eliminated shows a streak at the point of division.

Partial destruction may occur in a crescent along the lateral nail parts, appearing as if the nail had been scratched by an irregular body. Superficial destruction occurs on the undersurface, along the sides as well as the back of the nail.

TREATMENT OF NAIL PSORIASIS

Treatment of the more or less characteristic nail changes is, for the dermatologist, an arduous and thankless task. Local treatments in the author's own experience have generally not been satisfactory; tar and salicylic soap plasters often merely slow down further progress of the disease.

So far as local treatment for nail psoriasis is concerned, Dietel considers chrysarobin good but cignolin better. The latter is applied as a weak salve or with a brush. These soothing measures combine well with baths, and, after prolonged baths, good results are obtained from salicylic salves, using the cignolin salve only at night.

In psoriasis punctata of the nail plates, the use of mechanical appliances externally may be soothing and thinning to the nail plates. The patient himself may file them down as thin as possible. Instead of chrysarobin and similar salves or brushings, rubber plastic may be used, as it is much more convenient in ambulatory patients. The more or less active subungual hyperkeratoses occurring in outbreaks of psoriasis are amenable to certain measures, such as baths, highly concentrated salicylic salves, definite use of chrysarobin and cignolin preparations.

FIGURE 1. ATOPIC ONYCHIA. *Many transverse corrugations are present in the nail plates, a suggestive picture present in some atopic cases—the asthma, hayfever and eczema triad. This woman of 25 had had such a syndrome for many years with the nail changes shown for three years.*

FIGURE 2. INFECTIOUS ECZEMATOID DERMATITIS. *Bacterial infection of the periungual cutaneous surface is a weeping crusting eruption. The nail changes are secondary to this infectious process. A man of 32 has had an eruption on fingers and hand for four months' duration—closely located longitudinal ridging occurred one month after such infection.*

Whatever the character of nail psoriasis may be, it is so difficult to influence that Fuhs of Vienna considers x-ray treatment by all means the best. In some cases where constitutional and skin manifestations warrant, he advises x-ray therapy combined with arsenic in as high a dosage as practical. A 1 to 2 per cent sodium arsenate solution is injected *ad nates* with a 20 to 30 per cent total injection. For psoriasis which is generalized or in nail cases, the larger series of x-rays of 100 to 150 r. of an average hard ray, filtered through 1 mm. aluminum, may be given three times in two weeks on the dorsal side of the diseased nail parts and as required in repeated series at six weeks' intervals. In milder cases, especially paronychia psoriatica, the Bucky-Grenz ray may be successfully substituted in dosages of 200 to 44 r. in 10 cm. FHD, 10 kv. max., 10 MA., applied several times in a two weeks' interval.

The most frequent form of nail psoriasis, psoriasis punctate unguium, comes in least frequently for treatment. Most patients overlook the condition until it is called to their attention. The condition, however, is usually so cosmetic in nature that the patients notice it early, and it is found that by shaving down the nail plates with the sharp edge of a microscope slide or a file the nails become more sightly. If the disease process continues in the nail matrix, forming new pits, local treatment with roentgen rays, grenz rays, and thorium-x-degea may influence them, but the application of pyrogallic acid, chrysarobin, or cignolin is not advised.

Psoriatic weakening in the lunula, or "half-moon" areas, papules in the nail bed and under the free edges, as well as subungual hyperkeratoses, with decided elevation and destruction of the nail plates, put an end to occupational usefulness.

X-ray treatments should not be used in acute cases, as they are not protected from relapse and the rays have only a limited area of application.

HYPOVITAMINOSIS

Nail disturbances due either to lack of vitamins or improper usage of the vitamins is now a well-recognized clinical condition. Mild vitamin deficiency can occur in people without causing any real marked symptoms, as shown by McLester. The nail cases which show bodily lack of vitamins are characterized by transverse depressions with longitudinal striations, which in very severe instances advance to nail destruction. Usually the vitamins B_2 and G are incriminated and ingestion of foods and medicaments containing these vitamins produces a clinical cure.

NAIL DISTURBANCES DUE TO CONTACT AND ASSOCIATION WITH ATOPIC DERMATITIS

Nail deformities due to underlying allergic eczemas comprise a well recognized clinical finding. Usually there are the transverse corrugations of the nails which tend to disappear as the new nails grow distally when the dermatosis is cured.

Under this heading may be put nail changes due to occupational dermatoses and irritation due to various irritants as cosmetics, etc. In occupational disturbances there are found variations from simple stains to real nail destruction.

The problem of cosmetics in respect to nail changes is far from being settled. Several cases have been observed where there is no question about the irritation of the nails' being due to nail polish, polish removers, and nail base preparations. However, the number of cases is very small compared to the large number of people who use these substances.

FIGURE 3. ONYCHOMYCOSIS (RINGWORM). *Fungus infection has taken place in all the nails with no glabrous skin involvement. The T. gypseum fungus was grown on culture. This man of 36 had noticed two years ago the typical finding of beginning ringworm infection. Powdery disintegration first involved the distal ends and borders with increasing areas involved until the somewhat heaped edges or site of more fungus invasion as shown in this case.*

FIGURE 4. PSORIASIS. *Hyperkeratosis of nail plate with subungual separation of plate with chalky white appearance. Male, 65, with psoriasis of glabrous skin for many years. Nail changes have been present for six years.*

TUMORS

Various growths can occur around the nails as elsewhere on the body. Angioma, melanoma, keratoses, malignancies of various types and, of course, ordinary verrucae do occur. The treatment of these various tumors is that of tumors elsewhere on the body.

For warts, it is well to use conservative treatment because of the possibility of scarring where warts are removed by electrothermic methods. Warts around the nails usually respond well to small doses of radium therapy. Bismuth injections and local mercurial ointments are used where the verrucae are multiple.

TRAUMATISMS

Nail deformities due to direct injury and to chemicals are well known. A condition known as fissured nails is found quite often and can only be

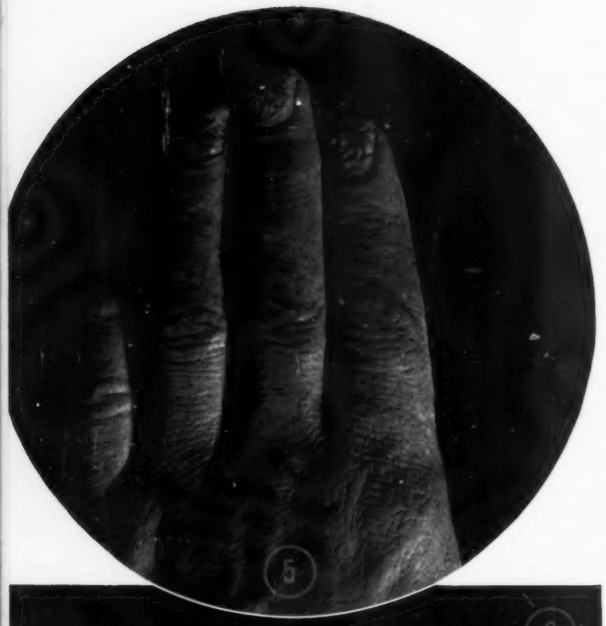


FIGURE 5. PSORIASIS. *Nail changes are usually punctate pitting. Woman, 36, has had psoriasis of glabrous skin for many years; the nails have been involved for three and one-half years.*

FIGURE 6. CONTACT ONYCHIA (NAIL BASE). *Marked transverse depressions with discoloration and some destruction occur in nail plate after application of certain lacquer nail base. This woman, 38, noticed blackish brown discoloration with beginning of plate depressions two weeks following one application of a lacquer base.*

FIGURE 7. CONTACT ONYCHIA (TRICHLORETHYLENE). *The hydrocarbons produce a distinctive blackish discoloration along with evidences of longitudinal ridging and punctate areas of destruction. A man of 56 had worked many years handling hydrocarbons of different types. These changes started four months preceding observation. There were no clinical signs of involvement of adjacent cutaneous areas.*

FIGURE 8. X-RAY DYSCRASIAS. *Repeated exposures to x-ray produce thinning and atrophy with discoloration. Associated cutaneous changes are present. This marked loss of portions of nail plate occurred in a physician who had performed a great many fluoroscopic examinations without rubber glove protection.*

remedied by suturing the fissured portions together.

MISCELLANEOUS

Many nail disturbances are found in various conditions. Some cases of the association of alopecia areata with coincident dystrophy of the nails have been described. In some instances they are probably two separate conditions; in others it is believed that they are related. The question of nail changes due to endocrine disturbances is debatable, but there are instances of definite nail changes in association with diseases of the thyroid gland. Nail changes have been observed in 2 recent cases of lichen planus. One was simply that of papules in the nail bed and another one, in a physician, showed marked nail destruction in association with a severe generalized lichen planus.

Tuberculosis has been found to produce a dactylitis and paronychia with concomitant changes in the nails. Biopsy is usually necessary to make an absolute diagnosis.

Many severe illnesses, in which there is a lowered nutrition, may cause the transverse furrows across the nails, and in some instances a complete loss of the nails themselves.

FIGURE 9. ARGYRIA OF NAILS *(From Neo-silvol). (Patient of Dr. Arthur C. Curtis, University of Michigan, Ann Arbor.)*

FIGURE 10. HYPOCHROMIC NORMOCYTIC ANEMIA. *Note multiple lamination of tips of nails. Woman 32 years. (Patient of Dr. Lourdes B. Gordon, Chicago.)*

FIGURE 11. FINGERNAIL CHANGES ASSOCIATED WITH HYPERTHYROIDISM. *Colored woman with hyperthyroidism presented by Dr. Francis E. Senear and (by invitation) Dr. R. H. Harris before Chicago Dermatological Society. A prominent Chicago thyroid surgeon notes that "fingernails look as if they have a sliver under them." Patient first observed that more repeated cleaning is necessary. Separation may be serrated. Inverted convexities may then develop.*

FIGURE 12. ONYCHOLYSIS. *With chronic arthritis. (Courtesy of Dr. Norman Tobias, St. Louis.)*

Case Report

Xanthoma of the Lung

WILLIAM B. FORD, CHARLES W. THOMPSON, AND BRIAN BLADES

GEORGE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON, D. C.

INTRATHORACIC xanthomas are rare. In 1937, Phillips[1] reported 2 cases of intrathoracic xanthomas and referred to 3 similar lesions described in the literature. All of these neoplasms were located in the costovertebral angle. In our clinic we have removed one xanthoma of the posterior mediastinum. Only one true xanthoma of the lung has been recorded in the world literature.[2] The rarity of this lesion justifies the recording of a second case.

Xanthomas are loosely encapsulated, golden yellow tumors. The characteristic foam cell, which is found on microscopic examination, was not described until 1892. Since that time there have been many theories advanced as to the exact origin of these neoplasms. Both connective tissue and fat have been considered as the anlage for xanthomas and Virchow[3] is of the opinion that these tumors develop from a combination of the two. The characteristic foam cell is often absent unless the lesion is in or about a tendon sheath.

CASE REPORT

A 35-year-old white woman was found to have a well circumscribed opaque shadow, approximately 2.5 cm. in diameter, in the left lung during the course of a chest survey in connection with tuberculosis control (Figure 1).

From the Departments of Medicine and Surgery, The George Washington University School of Medicine, Washington, D. C.

An investigation of her past history revealed that six years before (Figure 2) an indistinct radiopaque shadow had been discovered in an x-ray film of the chest and she had been advised to have roentgenograms of the chest at six-month intervals. Since there was no appreciable change in the size of the mass, active treatment was not undertaken. Sputum examinations and cultures were negative for acid-fast bacilli.

The patient had had the usual childhood diseases with good recovery and no history of other disease was given.

The family history was significant because her father had died of pulmonary tuberculosis, but was otherwise not helpful.

Physical examination revealed a well-developed female. Temperature, pulse, and respirations were within normal limits and there were no significant findings.

Examination with the fluoroscope revealed normal excursion of both diaphragms and the well circumscribed mass in the left lung. Roentgen localization of this mass placed it in the lingula of the left upper lobe.

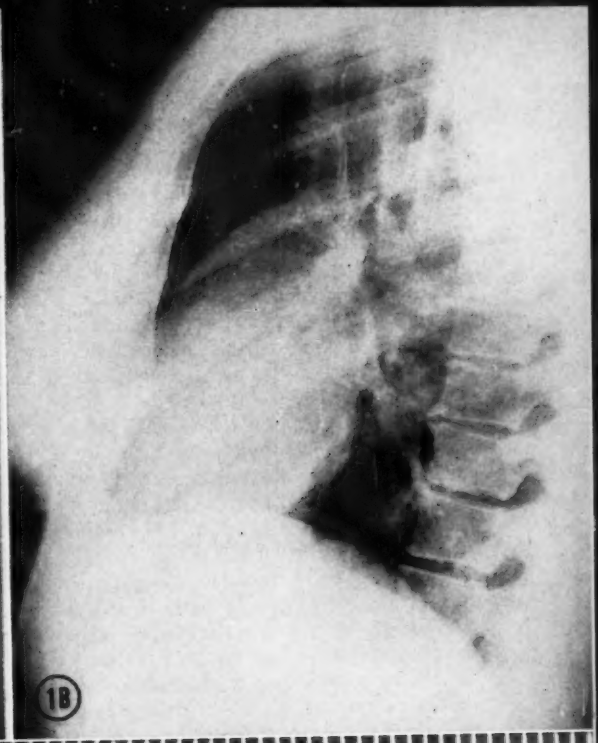
Operation—Exploration of the left thorax re-

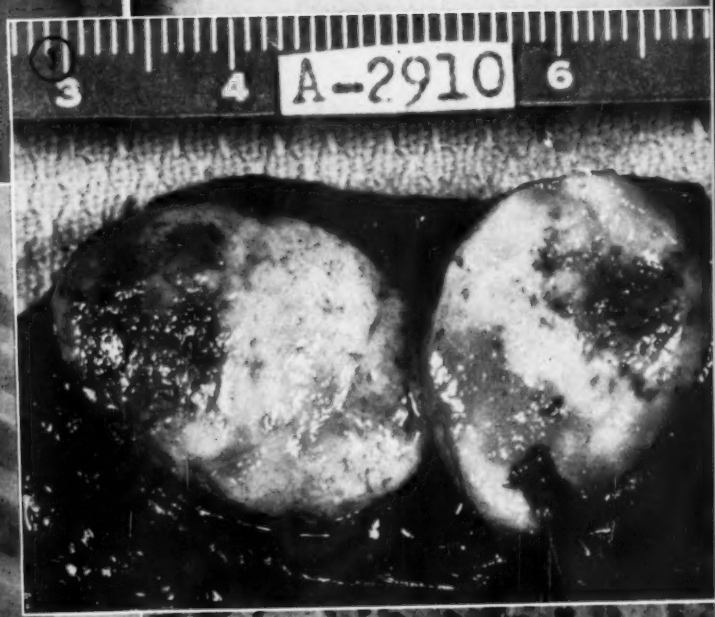
FIGURE 1. *A.* Frontal projection demonstrating a well circumscribed mass in left lung. *B.* Lateral view localizing tumor in lingula of the left upper lobe.

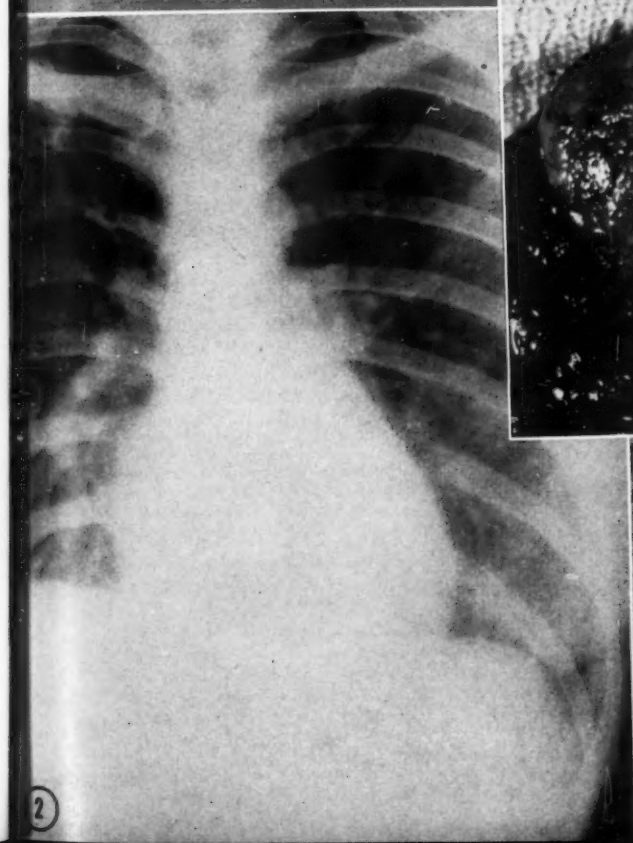
FIGURE 2. Roentgenogram of chest approximately six years before operation.

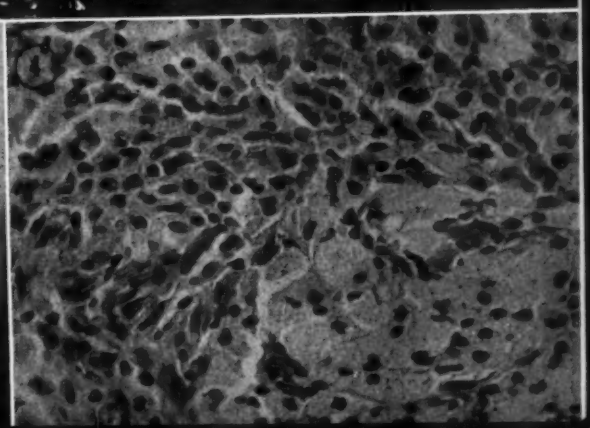
FIGURE 3. Gross appearance of tumor.

FIGURE 4. Microscopic section showing characteristic foam cells.


1B


3
4
A-2910
6


2



vealed an easily palpable, visible mass involving the lingula and, accordingly, a lingulectomy was performed (B.B.) (Figure 3). The gross appearance of the lesion suggested xanthoma since the tissue had a golden yellow appearance with a few hemorrhagic foci throughout. The mass measured 2.5 cm. in diameter and the parenchyma of the lung immediately adjacent to the mass was partially atelectatic.

Microscopic examination—The tumor was separated from adjacent lung tissue by a thin fibrous capsule containing a mild lymphocytic infiltration. It was composed of irregular spindle cells interspaced with variously sized clusters of typical foam cells having small round nuclei and large, pale, vacuolated cytoplasms which stained intensely with Scharlach red. Plasma cells in varying numbers were found throughout the sections, as well as were small, local collections of lymphocytes. Scattered Touton giant cells were present. Kassen trichrome stains failed to reveal the presence of any significant collagenous stroma. The blood vessels were small and they consisted of endothelial-lined spaces (Figure 4).

Diagnosis—Xanthoma of lung.

The postoperative course was entirely uneventful and the patient left the hospital in a week. During her postoperative stay a complete skeletal survey was entirely negative for other tumors.

SUMMARY

A case of solitary pulmonary xanthoma treated by surgical extirpation has been reported. A review of the literature has shown that this is the second case of this nature to be recorded.

It is important to note that in this case the original recommendation was to observe the tumor by frequent roentgenologic examinations and surgical intervention was not considered. Since it is impossible to make a precise differential diagnosis between xanthoma, adenoma, and even an indolent, slow-growing squamous cell carcinoma of the lung, this advice was dangerous. We have recently performed a pneumonectomy for bronchiogenic carcinoma in a patient who had a demonstrable lesion in the right upper lobe for six years. Roentgenograms of the chest were taken at frequent intervals, but since there were no symptoms surgical intervention was not recommended. It was not until the patient had hemoptysis that treatment was instituted. The carcinoma could be removed, but the patient died of metastasis within a few months.

Until improved methods of diagnosis are perfected to establish without doubt the nature of a mass in the lung, surgical excision offers the safest method for both diagnosis and treatment, provided the patient's general condition does not preclude a major surgical procedure.

REFERENCES

1. Phillips, Edgar W.: J. Thoracic Surg. 7:74-95, 1937.
2. Scott, William H., Jr., Morrow, Andrew G., and Payne, T. P. B.: J. Thoracic Surg. 17:821-825, 1948.
3. Virchow (Quoted by Ewing, James): Neoplastic Diseases. Philadelphia, W. B. Saunders Company, 1940.

COMING POSTGRADUATE COURSES

All-Austrian Congress of Physicians, Salzburg, Austria

Arranged by the Austrian Van Swieten Society for promotion of medical research and refresher courses for physicians, at 32 Schwarzstrasse, Mozarteum-Salzburg, September 7-9. Principal subject will be "The vegetative nervous system and its relations to pathogenesis." Lecturers include Professors Hoff, Erlacher, and Antoine, all of Vienna; Professor Mark, Rostock; Prof. Sunder-Plassmann, Muenster; Prof. Bing, Baltimore.

University of Minnesota Medical School, Minneapolis

Continuation course in neurologic roentgenology, at the Center for Continuation Study, October 30-November 4. Emphasis will be placed on different technics available for roentgen examination of the central nervous system, and on correlation of clinical and roentgenologic findings. Presentations on anatomy and pathology of the central nervous system will be given early in the course. Visiting faculty members of the course include Drs. Arthur E. Childe, Winnipeg; Philip J. Hodes, Philadelphia; Dabney Kerr, Iowa City; and Knut Lindblom, Stockholm, Sweden.

GENERAL PRACTICE *Review*

To provide the general practitioner, as well as the specialist with concise, readily available information on the latest, proved methods of treatment of conditions commonly encountered in an average practice, POSTGRADUATE MEDICINE *offers this department to its readers. It is not, of course, intended to present these discussions as the only acceptable therapeutic procedures to be used, but rather to offer simple regimens and recommendations based on the extensive experience of the physicians who prepared these summaries.*

Treatment of Erysipelas

MAURICE J. COSTELLO

NEW YORK UNIVERSITY COLLEGE OF MEDICINE AND THIRD MEDICAL DIVISION,
NEW YORK UNIVERSITY-BELLEVUE MEDICAL CENTER

THE basic treatment of erysipelas has become simplified to a great extent by the introduction of the sulfonamide drugs and by the administration of penicillin by injection. Before the use of these drugs, there were always about a dozen cases of erysipelas in the dermatologic wards at Bellevue Hospital. Today there rarely is seen a single case which has passed the gauntlet of effective therapy outside the hospital, or on the other inservices.

Penicillin is preferred to the sulfonamide drugs because of the fewer and less serious side effects accompanying the former. Since the patient is usually confined to his bed in a hospital or in his home, aqueous penicillin in relatively large doses is preferable to the penicillin in oil preparations. A dosage of 100,000 units in water or normal saline is injected intramuscularly every three hours (eight times a day) for three to five days.

Penicillin in oil, crystalline procaine penicillin G suspended in peanut oil, containing 2 per cent aluminum monostearate 300,000 to 400,000 units twice daily is also very effective, but less rapid in its therapeutic effect than aqueous penicillin. The dosage of penicillin is reduced for children. In feeble adults or weak children, it may be advisable to continue therapy with these effective drugs to prevent relapse and complications, especially bronchopneumonia and cardiac failure in the aged.

The sulfonamide drugs are also very effective, but they are more toxic than penicillin, and should be administered with greater care. Sulfathiazole and sulfadiazine are probably the most effective and least toxic. In adults, if the drug is well tolerated, the dose is 1 gm. every four hours for at least three, and preferably five days. Adequate alkaline therapy such as bicarbonate of soda 10 gr. every four hours and increased fluid intake are important in proper treatment. Urinalyses and daily blood counts are advisable to guard against renal irritation and significant changes in the blood count. However, secondary anemia and leukocytosis are the rule in erysipelas.

The fall of temperature and the amelioration of symptoms occur within forty-eight hours. Infants in the first year of life may be given 2 gr. (0.12 gm.) to 10 gr. (0.3 gm.) three times a day. Children 1 to 6 years of age may be given 10 gr. (0.6 gm.) three times a day; children over 6 years 15 gr. (1 gm.) three times a day.

Rest in bed is essential. Local therapy should consist of cold wet dressings of saturated boric acid solution, magnesium sulfate, normal saline wet dressings, or 1:8000 potassium permanganate compresses. Penicillin ointment 1,000 units to the gram and bacitracin ointment are of value when applied locally.

Roentgen rays, the ultraviolet light, and erysipelas antitoxin are no longer used in treatment of erysipelas. Andrews, in a personal experience

with chronic recurrent erysipelas, advises injections of streptococcus erysipelatous toxin for recurrent erysipelas.[1]

One hundred and six patients suffering from erysipelas were treated with the sulfonamide drugs on the dermatologic wards of Bellevue Hospital.[2] One hundred and two were discharged from the hospital cured, 2 patients with recurrent erysipelas were unimproved, and 2 debilitated patients died. One patient developed an abscess which had to be incised and drained.

Patients suffering from repeated attacks of erysipelas may develop chronic lymph stasis or so-called solid edema or lymphedema. In these patients, search should be made for foci of infection in the sinuses, teeth, tonsils and the sulfonamides mentioned should be administered over a relatively long period of time. Sulfapyridine, although one of the most toxic sulfonamide drugs, may prove superior to the others in the treatment of chronic pyogenic infections.

Aureomycin is said to be effective if the causative organism of erysipelas is sensitive to this drug. The effective dose is 500 mg. four times a day. Experience with streptomycin is limited because of the superior therapeutic results obtained with the other drugs.

Plastic surgery has proved to be of value in patients suffering with chronic lymphedema, especially when this condition affects the eyelids, ears, lips, genitals, and extremities.

The prophylactic therapy of erysipelas consists of prevention, prompt recognition, and immediate treatment of scratches, abrasions, ulcers, and wounds which serve as avenues of infection. Erysipelas is not highly infectious, but physicians and nurses caring for patients with erysipelas should not attend women in confinement or assist at surgery operations.

Erysipeloid (Rosenbach) caused by the Erysipelothrix of swine erysipelas which occurs as tense, glistening, erythematous dusky patches principally on the fingers and hands is not to be confused with erysipelas, and erysipelatous eruptions or erysipelas-like eruptions resembling erysipelas should not be referred to as erysipeloid. Erysipeloid responds to treatment with the sulfonamides and penicillin in the same dose and time interval described for erysipelas.[3]

REFERENCES

1. Andrews, George C.: Diseases of the Skin. Ed. 3. W. B. Saunders Company, Philadelphia and London; page 278.
2. Costello, Rubinowitz, and Landy: New York State J. Med. 42: (December 15) 1942.
3. Costello, M. J.: Arch. Dermat. & Syph. 52:400 (November-December) 1945.

Management of Food Allergy

ALBERT H. ROWE

OAKLAND, CALIFORNIA

Foods or inhalants, or both, according to this writer's experience,[1] are the usual causes of clinical allergy, due to blood-borne allergens. Drugs given by mouth or injection and infectants are much less frequent causes.

Food allergy unfortunately has received inadequate recognition, partly due to unwarranted dependence on skin testing, whose fallibility in diagnosis[2] long has been recognized, and also to the failure to remember the many manifestations of clinical allergy resulting from food allergy. Only with an adequate trial diet can food allergy be studied and gradually controlled.

The frequency of food allergy and other causes of clinical allergy in order of relative importance are indicated in Table 1. These data have been determined by the accurate and adequate use of the writer's standardized elimination diets during the last twenty-five years. Skepticism of this role of food allergy is not justified by any physician who has not used these elimination diets or any other successful methods of diet trial[3,4] accurately for advised periods in many patients. Much chronic invalidism and many unwarranted diagnoses of functional and psychosomatic disease occur as a result of this failure to consider and control chronic food allergy.

CHARACTERISTICS OF FOOD ALLERGY

1. Food allergy may occur at any time of life, even in old age.

TABLE 1

MANIFESTATIONS OF CLINICAL ALLERGY

TYPES OF ALLERGY RESPONSIBLE IN ORDER OF PROBABLE FREQUENCY	Foods	Inhalants	Drugs Sera	Infectants
1. Bronchial asthma, allergic bronchitis, and croup	1	1	2	4 rare
2. Nasal allergy (hay fever, recurrent colds, postnasal, mucus, recurrent sore throats, or laryngitis).				
a. Seasonal (any season, especially spring to winter)	3	1	4	4
b. Perennial	2	1	4	4
3. Atopic dermatitis	2	1	4	4
4. Urticaria and angioneurotic edema	1	1	2	4
5. Acne (lipoidosis with or without infection the major cause). Possible allergy too	3	?	2	?
6. Contact dermatitis (epidermal cell sensitized)				
7. Gastrointestinal allergy (canker and recurrent cold sores, oral and pharyngeal inflammation, distention, pyrosis, nausea, vomiting, cramping, pain, peptic ulcer, regional enteritis, diarrhea, constipation, mucous and chronic ulcerative colitis, irritable bowel, proctitis, pruritus ani, require study of possible allergy)	1	3	3	4?
8. Migraine, sick headaches, cyclic vomiting, bilious attacks (neuralgia, paresis, confusion, and idiopathic epilepsy, at times due to allergy)	1	3	4?	4?
9. Allergic toxemia (fatigue, bodily aching, confusion, etc.)	1	3	4	4?
10. Arthritis and synovitis (arthralgia, swellings of joints and tendons, rheumatic fever, rheumatoid arthritis, intermittent hydrarthrosis)	1	4	3	2
11. Ocular allergy (lid dermatitis, blepharitis, conjunctivitis, iritis, corneal ulcers, cataracts, retinal allergy, ocular pain)	3	1	2	3
12. Aural allergy (dermatitis of ear and ear canal, edema of Eustachian tubes, deafness, tinnitus, vertigo, dizziness)	1	2	3	4?
13. Urogenital allergy (allergic cystitis, spasm in bladder and ureter, vulval canker-like ulcers, inflammation and dermatitis, menstrual pain, leukorrhea)	1	3	3	4?
14. Cardiovascular allergy (cardiac irregularities, psuedo-angina, coronary thrombosis, thromboangiitis obliterans, periarteritis nodosa, erythema nodosum and multiforme, glomerulonephritis)	3	4?	1	1
15. Allergic purpura, agranulocytosis, and bleeding	2	4?	1	4?
16. Allergic fever (recurrent or intermittent)	1	3	1	4?
17. Allergy to Rh substance (erythroblastosis fetalis, transfusion reactions)				

2. Skin testing by the scratch test usually gives large positive reactions to those foods causing marked immediate symptoms, and negative or slight reactions when cumulative or chronic symptoms result. Positive reactions too often are due to potential allergy or nonspecific causes.

3. A seasonal exaggeration of food allergy often occurs from early fall to late spring, especially in the winter.[5] Symptoms may diminish or disappear in the summer. Food allergy also may decrease or disappear in dry, inland areas.[5]

4. Food allergy produces recurrent attacks,[1,7] usually at regular intervals, more often than do inhalant and the infrequent bacterial allergies. The exhaustion of the reacting bodies to the causative foods during the attacks and their gradual reaccumulation up to a reacting level explain why the allergenic foods can be eaten between attacks with few or no symptoms.

5. It may cause localized reactivity in one area of a given tissue as the skin, and may affect one or several tissues in the same patient.

6. Fever may be due to food allergy[6] as well as to drug or serum allergy.

MANIFESTATIONS OF FOOD ALLERGY

Space permits only brief discussion of the manifestations and treatment of food allergy.

The writer's good and excellent results in a recent series of 1,500 cooperating patients with bronchial asthma indicated that food allergy was the sole cause in 40 per cent up to the age of 5

years,[7] and in 20 to 40 per cent in the other age groups, even over 55 years.[9] Inhalant allergy with or without food allergy was equally important and infectant allergy was a rare secondary and a rarer sole cause. Drug allergy occasionally was found to be responsible.

The following typical history of asthma in children due to food allergy has been described by this writer:[7] Eczema with or without frequent colds may occur in the first year or two. Then regularly recurrent asthma lasting two to five days with or without fever, nausea, and vomiting develops from early fall to late spring, preceded or accompanied by nasal symptoms. The regularity of the attacks and the decreased or absent symptoms in the summer have already been explained. Their recurrence or exaggeration in the early fall to winter, especially on return to school, wrongly suggests respiratory infection rather than food allergy as the cause. In fact, regularly recurring "head colds" with or without cough, wheezing, and fever are due usually to food allergy rather than to infection.

Nasal-sino-pharyngeal perennial symptoms also require equal consideration of food and inhalant allergy. Thereby the rarity of bacterial allergy becomes evident. Surgery rarely is indicated except for removal of polyps, which are usually due to chronic food allergy.

This writer long has realized the frequency of gastrointestinal allergy.[11,12] Any area from the mouth to the anus may be involved. Food allergy is dominant though inhalant and drug allergies may produce symptoms. Canker sores usually result from food allergy. Gastric allergy produces epigastric and substernal heaviness, pain, pyrosis, belching, nausea, vomiting, and other symptoms. Food allergy is important as a cause of unexplained pain in any part of the abdomen, especially in the lower and upper right quadrants and in the epigastrium. Inflammation, edema, smooth muscle spasm, and other tissue reactions also may occur in any part of the small intestine. Regional enteritis[18] has been controlled by the elimination of allergenic foods by this writer. Evidence favors allergy especially to foods as the usual cause of the irritable bowel, mucous colitis, diarrhea, proctitis, and pruritus ani, at times of constipation and especially of chronic ulcerative colitis.[13,14] With adequate and experienced study of allergy the writer has found functional and psychosomatic causes infrequent.

Similar study has shown the indicated frequency of food and other allergies as the causes of cutaneous, ocular, aural, urogenital, joint, and cardiovascular allergies, of allergic purpura, recurrent headaches and migraine, and allergic fever.[1,11] Allergic toxemia[15,16] characterized by fatigue, aching in joints and body, confusion, dopiness, depression, and other symptoms are usually due to food allergy.

DIAGNOSIS AND TREATMENT

All patients must have a carefully taken history, thorough physical examination, and indicated laboratory and roentgenologic studies to discover all possible disease. The dietary history[1] especially must record all food dislikes and disagreements, which may indicate specific allergies. Environmental and drug histories are also necessary. As shown in Table 1, inhalant and to lesser extents drug and infectant allergies always need consideration and indicated control. Only the control of food allergy, however, will be discussed in this paper.

A trial diet must be used to study possible food allergy because of the above discussed fallibility of the skin test. The advantages of the elimination diets[1] are their exclusion of foods which frequently cause allergy, their printed lists of allowed foods, detailed menus and recipes for bakery products, and finally the maintenance of weight and nutrition for brief or prolonged periods if they are accurately prepared and followed.

Recognition of food allergy in bronchial and nasal allergy has depended especially on the cereal-free elimination diet.[1] This also is used in atopic dermatitis, migraine and recurrent headaches, possible allergic fever, allergic toxemia, ocular and aural allergy. The fruit-free elimination diets are usually used in studying food allergy in gastrointestinal and urogenital tracts, in the joints, in urticaria, and angioneurotic edema and in any other problem in which allergy to one, more, or even all fruits is suspected. If symptoms are not relieved with adequate trial, then foods in the diet may be suspected, and other supplemental or minimal diets are utilized. At all times, the diet with calcium and vitamins as advised elsewhere[1] must maintain nutrition and a satisfactory weight.

The diet must be continued until the symptoms have been absent for two to three times as long as usual periods of former relief. Thus if daily symptoms are relieved for five to seven days, or if symptoms recurring every two weeks are absent for four weeks, other foods, one daily for four to five days, can be added, eliminating any which reproduce symptoms.

When elimination diets are prescribed the patient must receive the list of allowed foods, the published detailed menus, and recipes for bakery products,[1] and the physician must discuss the reasons for the preparation of the diet with him. A list of forbidden foods, especially without menus or recipes for bakery products, must never be given.

In the cereal-free elimination diet for infants,[1] soy bean milk such as Mull Soy, Soy-o-lac, or Sobee is substituted for animal milks. If allergy or intolerance for soy bean exists, the writer's strained meat formula, using Gerber's or Swift's strained meats, is utilized. A similar formula with more strained meat, containing 100 to 140 gm. of protein and

over 2,000 calories in each 2,000 cc. has been used successfully in the control of food allergy in chronic ulcerative colitis.[14] It is preferable to protein hydrolysates because of its palatability. Studies by Dr. Cannon[17] have shown that soy bean is not a complete protein. Hence supplemental meat in infants and children is desirable.

With the prolonged total elimination of allergenic foods, tolerance may return. Children often regain complete or partial tolerance with such exclusions in one to three years. Such restored tolerance is less frequent in adults. This writer has failed to establish tolerance by oral or hypodermic administration of food allergens. It is probable that apparent results have occurred in nonsensitive patients whose food allergy was assumed from positive skin reactions alone.

While the causative foods are being determined by elimination diets, the following drugs may be utilized to control symptoms: Epinephrine hypodermically and by inhalation is of greatest value in bronchial asthma; ephedrine with or without barbitals will help some mild symptoms; aminophylline by mouth, rectum, and vein is important in severe bronchial asthma. The antihistaminic drugs have benefited urticaria and some nasal and bronchial allergy due to food allergy. They are not used for the control of other manifestations of food allergy because of their relative ineffectiveness and their side reactions.

REFERENCES

1. Rowe, A. H.: Elimination Diets and the Patients' Allergies. Philadelphia, Lea & Febiger, 1944.
2. ———: The evaluation of the skin reactions in food sensitive patients. J. Allergy 5:135, 1934.
3. Rinkel, H. J.: Food allergy. II. The technique and clinical application of individual feeding tests. Ann. Allergy 2:504, 1944.
4. Randolph, T.: Food allergy. M. Clin. North America, January, 1948.
5. Rowe, A. H.: Seasonal and geographic influence on food allergy. J. Allergy 13:55, 1941.
6. ———: Fever due to food allergy. Ann. Allergy 6:252, 1948.
7. ———: Bronchial asthma in infants and children. California Med. 69, No. 4, October, 1948.
8. ———: Bronchial asthma in adult life, from the ages of 15 to 55 years. (To be published.)
9. ———: Asthma in patients over the age of 55. Ann. Allergy 5:509, 518 (November-December) 1947.
10. ———: Nasal allergy. Arch. Otolaryng. 28:98-195 (July) 1938.
11. ———: Clinical Allergy. Philadelphia, Lea & Febiger, 1947.
12. ———: Gastro-intestinal food allergy. J.A.M.A. 97:1440, 1931; Journal-Lancet 56:120, 1936.
13. ———: Chronic ulcerative colitis—allergy in its etiology. Ann. Int. Med. 17, No. 1, July, 1942.
14. ———: Chronic ulcerative colitis—an allergic disease. Ann. Allergy, November, 1949.
15. ———: Allergic toxaemia and migraine due to food allergy. California Med. 33:785, 1934; allergic toxemia and fatigue. Ann. Allergy 8:72, 1950.
16. Randolph, T.: Fatigue and weakness of allergic origin (allergic toxaemia) to be differentiated from nervous fatigue or neurasthenia. Ann. Allergy 3:418, 1945.
17. Cannon, P. R., Wissler, R. W., Steffee, C. H., Woolridge, R. L., and Benditt, E. P.: The evaluation of protein hydrolysates. J. Am. Dietet. A. 23, No. 10, October, 1947.
18. Rowe, A. H., et al.: Regional enteritis due to food allergy. (To be published.)

Treatment of Air Sickness

HARRY G. ARMSTRONG

MAJOR GENERAL, THE SURGEON GENERAL, UNITED STATES AIR FORCE, WASHINGTON, D. C.

GENERAL MEASURES

The treatment of airsickness[1] is directed principally to its prevention, for once the disease becomes established, its cure during flight is usually impossible. Bearing in mind the fact that airsickness is caused principally by overstimulation of the labyrinth from repeated vertical accelerations of the aircraft as it flys through "bumpy" air (or from aerobatics) and to a lesser extent from psychic influences, it is obvious that treatment must be directed to these two influences.

Since the number of persons who become airsick is roughly proportional to the amount and also the duration of accelerations experienced in flight, it follows that much can be accomplished through the proper operation of the airplane. Aerobatics should not be indulged in nor should sudden turns, climbs, or descents be made. Rough or "bumpy" air should be avoided during flight although this is not always possible.

A reclining posture with the face pointing upward minimizes stimulation of the sensory hairs of the utricles by the otoliths. This both helps to prevent airsickness and tends to relieve symptoms which have developed.

Gastric disturbances existing prior to flight, espe-

cially those due to overindulgence in food or drink or the use of cathartics, predispose to airsickness. Being hungry predisposes one to airsickness also and a moderate meal an hour or so before take-off is beneficial.

Those who think they are going to be airsick usually become so. Anxiety, grief, and fear seem to have an adverse effect as do bad odors, poor ventilation, and too warm a cabin temperature.

Keeping the mind occupied, reassurance, and keeping the eyes fixed on definite objects on the ground below are all good prophylactic measures.

SPECIFIC THERAPY

The best known drug for the prevention of airsickness is **hyoscine hydrobromide.** A dose of 1/100 gr. (0.65 mg.) by mouth should be taken about one hour before flight and repeated two hours later if considered necessary.

Recently Dramamine (β-dimethylaminoethyl benzohydryl ether 8-chlorotheophyllinate) has been reported as being very effective in the prevention and treatment of seasickness by Gay and Carliner.[2] However, Strickland and Hahn[3] in an extensive investigation of this same drug in the prevention or airsickness have not found it superior to hyoscine hydrobromide and found also that it possesses more undesirable side effects. These side effects consist of central nervous system depression leading to drowsiness and mental depression, dryness of the mouth, and in some cases, vertigo. The dose of Dramamine is 1½ gr. (100 mg.) by mouth thirty to sixty minutes before a flight.

In the Strickland and Hahn series, 28.7 per cent of the experimental subjects became airsick as evidenced by vomiting while 55.6 per cent of the controls who received a placebo became manifestly ill.

Airsickness is a self limited disease which normally does not persist for more than twenty minutes to one hour after the termination of a flight although in rare cases slight vertigo, nausea, and generalized weakness may persist for as long as twenty-four hours. There is no record of airsickness resulting in a fatality.

REFERENCES

1. Armstrong, H.: Principles and Practice of Aviation Medicine. Baltimore, Williams and Wilkins, 1943.
2. Gay, L., and Carliner, P.: The prevention and treatment of motion sickness. Science 109:359, 1949.
3. Strickland, B., and Hahn, G.: The effectiveness of Dramamine in the prevention of airsickness. Science 109:359, 1949.

Necrotizing Ulcerative Gingivitis

JOHN R. WILSON, D.D.S.

COLLEGE OF DENTISTRY, THE OHIO STATE UNIVERSITY, COLUMBUS

Vincent's infection (necrotizing ulcerative gingivitis) is a bacterial disease in which susceptible conditions are necessary before the organisms can invade. Spirochete and fusiform bacilli are described as the symbiotic organisms of Vincent's infection, and are always found in the acute stage of the disease. Usually present in the lesions with the spirochetes and fusiforms are vibrios, spirilla, cocci, and actinomycetes. The presence in the mouth of these organisms, usually present in Vincent's disease, is not conclusive for diagnosis and should be used only to confirm clinical diagnosis. Attempts at direct transmission from acutely infected to healthy mouths have been unsuccessful and tend to disprove the communicability of the disease.

Recognition of the predisposing factors is a requisite for complete, successful treatment. Some of the conditions which may lower the resistance of the tissues or present avenues of infection are overhanging margins of restorations, poorly contoured restorations, impinging clasps or bands, poor contacts allowing food impaction, drugs, trauma, or any predisposing periodontal condition, such as third molar flaps, or preexisting periodontal pockets in any part of the mouth. When teeth are not present Vincent's infection is found only when a restoration forms a pocket with the tissue.

Systemic factors may also prepare the tissue for infection. Any oral or extra oral disease may lower the resistance of the tissues and offer an opportunity for invasion. An infection of the tonsil may

also progress upward to susceptible areas around the teeth.

Many methods of treatment have been recommended for Vincent's infection. The acute condition usually responds to most types of therapy, providing there is debridement of the gangrenous tissue and other filth and debris including calculus. Thorough debridement and cleaning is the outstanding feature of successful treatment.

The treatment may be divided into two major steps; first, the treatment of the acute phase of the disease, and second, the elimination of the incubation zones. The treatment may also be divided into *office treatment and home care*. Both are equally important.

An outline of treatment is as follows:

First visit—Spray the mouth vigorously with 3 per cent hydrogen peroxide solution to remove mechanically the debris and pseudomembrane. This should be done two or three times. After spraying, the patient should retain the solution in the mouth and flush it between the teeth for one minute before emptying the mouth. The mouth should then be rinsed with water and the tissue should be dried and isolated with cotton rolls. Mapharsen or Chlorarsen (0.40 to 0.060 gm) should then be applied locally. The powder should be dissolved in 10 cc. of water and applied to the gingival sulci or pockets by use of a syringe with a blunt needle.

Home therapy—Rinse mouth with equal parts of 3 per cent hydrogen peroxide solution and warm water every two hours, forcing the solution between the teeth. Use a diet of milk, eggnogs, fresh orange and other fruit juices, and broths. Drink plenty of water. No hard, hot, coarse, spicy, starchy, or fried foods should be eaten. The patient should not smoke or use alcohol and should rest as much as possible. Toothbrushing should be stopped until advised to continue. In acute cases, penicillin chewing troches (20,000 units) are prescribed to be chewed for two hours after each meal three times a day. The chewing troche is used because it promotes a more prolonged penicillin level and also because it affords a method for mechanically cleansing and debriding the crown surfaces of the teeth.

Second visit—The therapy of the first visit should be repeated and any gross calculus should be removed. If acute symptoms have subsided, the penicillin may be discontinued and hydrogen peroxide water lavage reduced to three times per day. Instructions in home care should be instituted.

Third and subsequent visits—The hydrogen peroxide and Mapharsen or Chlorarsen therapy should be repeated and prophylactic procedures continued until all irritants are removed. All overhanging margins, rough margins, and impinging clasps should be eliminated. A full series of roentgenograms should be taken, and all incubation zones such as third molar flaps, or periodontal pockets should be removed. The occlusion should be balanced. Failure to continue the debridement process by not completely eliminating periodontal pockets and other incubation zones is responsible for most recurrences of this disease.

Management of Appendicitis

FREDERICK FITZHERBERT BOYCE

TULANE UNIVERSITY OF LOUISIANA SCHOOL OF MEDICINE, NEW ORLEANS

ACUTE appendicitis is a disease which permits positive, unqualified statements concerning its therapy. The treatment is surgical, first, last, all the time, and, in the opinion of most surgeons, at any time in his illness that the patient is first seen. Laparotomy should be performed as soon as the diagnosis is made, and performed on reasonable suspicion if a positive diagnosis cannot be promptly established.

In uncomplicated acute appendicitis the treatment is simple and there is no reason or excuse for trying to make it complicated. All that is required is appendectomy. The patient is practically always in good condition. He needs only routine preoperative preparation, and postoperative care involves little more than routine nursing care, with careful observation to make sure that unexpected complications are not overlooked because of complacency.

The wound is left undisturbed until sutures are removed, in the absence of indications to examine it earlier, and activity may be resumed promptly.

Some surgeons permit ambulation the day of operation. One warning is necessary—if the patient is suffering from concomitant intercurrent or constitutional disease, consultation with an internist or other appropriate specialist is always indicated.

The surgeon may follow his own preferences in respect to anesthesia, incision, and technic. Provided that these measures are correct in principle, technical considerations do not affect the end results in any way. Profound anesthesia is naturally undesirable at any time and is particularly dangerous in debilitated, elderly patients.

The general principles which should guide the surgeon in his procedure are (1) careful packing off of the remainder of the abdominal cavity from the field of operation; (2) abstinence from out-of-sight manipulations; (3) the use of a gentle, atraumatic technic; (4) complete hemostasis, and (5) as little handling of the intestine and other structures as possible. Equally good results have been obtained with a variety of methods of treating the appendiceal stump. There is no single best way.

The treatment of complicated or advanced acute appendicitis is also surgical but it has ceased to be simple. Space does not permit the discussion of the advantages of temporizing therapy versus immediate operation in ruptured appendices, appendiceal abscess, and peritonitis, but for a number of sound reasons I share the opinion of many experienced surgeons that delay in performing surgery is not justified:

1. The diagnosis of the exact stage of the pathologic process in the appendix is notoriously inaccurate and cannot be related to the clinical picture.

2. There is no certainty that localization will occur, or, if it has begun, that it will continue favorably; the localized processes may themselves rupture.

3. It is against all reason to argue that a properly conducted surgical procedure is as harmful as the continued entrance of infection into the peritoneal cavity from a perforated viscus.

In advanced cases of acute appendicitis a preoperative period of rehabilitation is frequently necessary, for the patient is sometimes so ill that otherwise the mere act of operation might be fatal. This period should be as brief as possible and should preferably not exceed a few hours. Laparotomy is then carried out, the surgical procedure being adapted to the conditions present. Whenever appendectomy can be done without undue risk, it should be done. Otherwise, simple drainage should be instituted. Drainage operations on the intestine are almost never warranted now that the far more effective method of constant intestinal decompression is available. Chemotherapeutic agents or other solutions are not placed in the peritoneal cavity.

Drainage is never instituted in simple unruptured acute appendicitis unless it is required because of troublesome oozing. The belief is growing that closure without drainage is also the wisest procedure even in ruptured appendicitis with purulent peritonitis, though the cavity should be well cleared (by suction) of purulent exudate before the wound is closed. Drainage of the abdominal wall may be justified if the appendix is gangrenous or has been ruptured in delivery and the wound has become contaminated.

Residual abscesses are drained according to the indications of the individual case. Subphrenic abscess is drained by an extraperitoneal procedure, to avoid crossing an uncontaminated pleural or peritoneal cavity. Pelvic abscess is usually satisfactorily handled by colpotomy or proctotomy. Drains should be so placed that they do not rest on, or lie among, loops of bowel or cross uncontaminated areas of peritoneum. The duration of drainage is best determined by the amount and character of the discharge and the patient's general condition.

In complicated acute appendicitis the most careful preoperative and postoperative management is necessary. The regimen should include sedation; control of distention and ileus by constant intestinal decompression (instituted before either becomes apparent); replacement therapy calculated on the basis of the needs of the special case and with a sparing use of salt solution; generous blood transfusions; oxygen therapy; and chemotherapy and antibiotic therapy, frequently by a combination of agents. A most important point concerning adjuvant therapy is that it should be continued until there is no doubt that the need for it is past; this is particularly true concerning chemotherapy and antibiotic therapy and intestinal decompression.

In conclusion, a word may be said about so-called **chronic appendicitis** and **recurrent appendicitis.** The surgeon who operates for the former condition would be well advised to assure himself that the symptoms which he proposes to relieve by appendectomy actually arise from the appendix.

In recurrent appendicitis, on the other hand, it is the surgeon's duty to warn his patient that operation is indicated and should not be delayed. It is also well to bear in mind that the patient does not have recurrent appendicitis until he has safely passed out of his current attack. In the same connection, both surgeon and patient must bear in mind that no time should be lost in removing the appendix if acute appendicitis has been treated expectantly or if only drainage has been possible at operation. Many patients have lost their lives in recurrent attacks because that policy has not been followed.

EDITORIALS

Postgraduate Medicine
OFFICIAL JOURNAL OF THE INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA — ORGANIZED IN 1915

Editor-in-chief: CHARLES W. MAYO, M.D.
Contributing Editor and Editorial Writer: MORRIS FISHBEIN, M.D.
Assistant Editor: SYLVIA COVET

JULY 1950

PROGRESS AGAINST POLIOMYELITIS

GRADUALLY infantile paralysis is changing in some of its fundamental characteristics. Once almost invariably a disease of the summer months, poliomyelitis now seems to be a year-round menace. Formerly, the disease was so characteristically a condition of the younger years that it was called infantile paralysis; now adults are being infected with increasing frequency. Perhaps also the infectivity of the condition is intensifying since the number of cases reported last year was the largest in history.

Formerly we believed that poliomyelitis was caused by a single specific virus. Research has established a multiplicity of viruses capable of producing the disease and these may be classified in types as are the pneumococci. Moreover, a number of related viruses seem capable of producing conditions resembling poliomyelitis but with much less virulence.

The possibility of developing protective vaccination has attracted the interest of many investigators and both chemical and physical technics have been devised for killing the virus; none of these technics is established as capable of producing a vaccine that will immunize. Research has concerned also the attempt to utilize an attenuated living vaccine but this work has not progressed sufficiently to indicate promise of virtue. Moreover, several competent virologists assert that there is little hope for an efficient vaccine within the next twenty years.

The attempt to control infantile paralysis by isolation and quarantine is doomed to disappointment because investigators have shown the widespread incidence of the virus. A city with a thousand cases of paralytic poliomyelitis will have as many as 200,000 people with virus in their bodies but without the disease. The virus they transmit through direct contact or by other means may cause paralytic poliomyelitis in another person. Indeed, the evidence now seems to be well established that the disease is spread in greater part by direct contact, but that insects such as the fly may occasionally transmit virus. There are no houseflies in Iceland yet there are outbreaks of poliomyelitis.

At this time children in great numbers are preparing to attend summer camps. Since infantile paralysis is most feared of childhood diseases, parents suffer great shock when they hear that a case of infantile paralysis has occurred in some summer

camp. Questions arise as to whether or not the camp should be closed and the children returned home where they may disseminate virus further; whether or not the child who is ill should be sent home; whether or not swimming should be permitted; whether or not camps should have a doctor in constant attendance.

Epidemiologists believe—at least the majority—that camps should not be closed when a case of the disease appears. The child who is ill should be placed in bed in a room to itself. Usual nursing care for contagious disease should be practiced. A competent physician should be summoned. Each child in the camp should have the temperature taken morning and evening; with any significant rise in temperature the child should be placed in the infirmary and brought to the attention of the physician.

Routine care of children with infantile paralysis now includes the application of hot wet packs in the acute stage of the disease. This contribution of Miss Elizabeth Kenny merits recognition. As early as possible every patient with paralytic poliomyelitis should have attention by a competent orthopedic surgeon, whose knowledge may make a great difference in the extent of ultimate permanent crippling. Whenever bulbar poliomyelitis occurs hospitalization is demanded and here the quality of constant attention may mean the difference between life and death.

The proceedings of the first international congress on poliomyelitis held in New York in 1948 provided a complete resume of every aspect of the disease. The proceedings have been published by Lippincott in book form and represent the most comprehensive information available. Another international congress is planned to meet in Copenhagen, in September, 1951, with symposia on many of the phases of the disease that are now undergoing investigation. Many pamphlets are also available to those who will write to the National Foundation for Infantile Paralysis, Inc., 120 Broadway, New York. Practically all significant advancement against the disease has come from research sponsored and aided by the National Foundation; its support of research and its dissemination of information have been carried forward on an international scale without regard to race, creed or color. Increasingly funds are being made available also for the maximum of mental and physical rehabilitation of those who have suffered from this disease.

MORRIS FISHBEIN

ATOMIC RESEARCH ON TOBACCO SMOKING

The use of tracers to discover what happens to various substances inside the human body is one of the most important technics in medical research. Recently a fellow in the Department of Pharmacology, University of Chicago, in a research supported by the Atomic Energy Commission traced the action of radioactive nicotine in the bodies of experimental animals.

Nicotine is made radioactive by an especially devised technic. Tobacco plants are grown indoors, in sealed glass containers, at the University "atomic farm." While they are growing, carbon dioxide containing radio carbon is pumped into the surrounding air. The plants take in the gas and eventually the radio carbon is attached to the various ingredients of the plants. Thus the nicotine in the tobacco was made radioactive. This nicotine was then injected below the skin of a rat and was promptly taken into the blood stream so that it appeared in the urine within a minute. The nicotine was traced by the use of the Geiger counter and the spectrophotometer.

Nicotine taken into the body tends to collect in the lungs, liver, heart, spleen, and kidneys. A rat excreted about 65 per cent of the nicotine it had absorbed within twenty-four hours. Much has been made of the effects of nicotine on the human heart. Studies made on the isolated heart of a guinea pig showed that the heart stops absorbing nicotine after a few minutes, in some manner protecting itself against too great an intake of this drug.

By the use of this technic, the investigator was able to detect amounts of nicotine as small as one-nine millionth of an ounce.

The research on nicotine was part of the extensive study now being conducted under the direction of Professor Eugene M. K. Geiling in the "atomic farm." Such drugs as digitalis, nicotine, and belladonna are now being made radioactive and are then traced through the body.

These new technics enable scientific medicine to extend knowledge in a way never before possible.

MORRIS FISHBEIN

THE NAMES OF PHARMACEUTICAL PRODUCTS

In the United States the Council on Pharmacy and Chemistry has given consideration to the names of pharmaceutical products since 1905. Products accepted for inclusion in New and Non-official Remedies have become known by the designation "N.N.R." to distinguish them from U.S.P. (United States Pharmacopeia) and N.F. (National Formulary) products. Recently the Scandinavian Pharmacopeia Council has agreed upon an "N.F.N." name for a long list of drugs included in the National Scandinavian Pharmacopeias or official formularies or addenda. The term "N.F.N." is abbreviation for Nordisk Farmakopenaevn. As far as possible N.F.N. names have been chosen which have already been included in the British or American Pharmacopoeia or which have been described as approved names for the British Pharmacopoeia Commission, or described as generic names for the Council on Pharmacy and Chemistry.

In *Acta Pharmacologica* for June, 1950, a complete list of the names approved for the Scandinavian countries is published together with the scientific names which indicate formula and the various trade names by which products are known as sold. Uniform endings have been chosen for products of various classes and there are uniform classifications to cover the form in which the product is administered. As the teaching in schools of pharmacy and of medicine will conform to this list another generation should see the names which are selected in common use.

A reference to the list shows how seriously manufacturers of pharmaceutical products concern themselves with names that have selling value. Here one finds desoxycorticosterone known as cortate, cortiron, doca, ocriten, and percorten. The new Scandinavian name for the antihistamine commonly called benadryl and known also as allergin, amidryl, benzhydryl, densentol, etc., will be diphenhydraminum. For each of the antihistamines there are apparently from two to six names and since there are more than twenty-four of these, the burden on the mind of the prospective physicians and pharmacists is considerable. The variations are legion as with testosteronum which is called testosterone, orchistin, neo-hombreol, perandren, textex, testin, testodrin, testoviron and virox.

Somewhat in the same direction are the activities of the Committee on International Pharmacopoeia of the World Health Organization. The first text prepared will soon be available and will be sent to various nations with a view to incorporation of standards and names in the many pharmacopoeias of the world. The World Medical Association has established a committee on the international pharmacopoeia so that the point of view of the physician as well as that of the pharmacist and pharmacologist may be represented in these actions.

MORRIS FISHBEIN

PARENTS AND THEIR CHILDREN

Any father or mother of a fair-sized family, or indeed of even one child, has frequently wondered whether or not the children really give a "whoop in Halifax" for them and whether or not the love that they were languishing on the progeny was worth even the moderate effort entailed. The psychologists and psychiatrists have

been greatly concerning themselves with this matter in recent years and venture the suggestion that parental attitudes of the past have possibly created as much delinquency as they prevented.

Dr. Freda Kehm[1] says that children should feel free to express their hostilities against other members of the family. If your little boy calls you a dope or a stinker or says that he hates you, refrain from socking him so that he falls across the room or even from reproving him. Just tell the little tyrant that you understand why he is angry but that you love him just the same. Make Mary feel that you really love her all the time although you may sometimes be greatly annoyed by what she does. If she has just tossed your best piece of old Delft china on the floor, Mama must not say that Mama will dislike Mary if she does that again. Instead, says Doctor Kehm, parents too should feel free to show their displeasure and should accept the fact that their children hate them at times.

Especially is the parental relationship to the handicapped child a serious problem. Again and again physicians see families in which the mother has lavished love and attention on the handicapped or subnormal baby with resultant frustration, bitterness and anxiety among those not handicapped.

Anger is a common emotional reaction but one that is more destructive than productive; intelligent people divert the reaction of anger into useful and less harmful actions. Loving mothers and fathers need to give thought to their relationships with their children with the realization that overprotection may be as dangerous as neglect.

MORRIS FISHBEIN

1. Freda Kehm, Ph.D.: Parents Are Teachers, Too. The Crippled Child 27:3 (April) 1950.

International Assembly—Chicago, November 6 to 9, 1950, 35th Year

INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA

OFFICERS OF THE ASSOCIATION

PRESIDENT
Dr. Waltman Walters, Rochester, Minn.

PRESIDENT-ELECT
Dr. Richard B. Cattell, Boston, Mass.

PRESIDENTS OF CLINICS
Dr. John F. Erdmann, New York, N. Y.
Dr. Frank H. Lahey, Boston, Mass.

MANAGING DIRECTOR
Dr. Arthur G. Sullivan, Madison, Wis.

SECRETARY
Dr. Tom B. Throckmorton, Des Moines, Ia.

TREASURER AND DIRECTOR OF FOUNDATION FUND
Dr. Henry G. Langworthy, Dubuque, Ia.

SPEAKER OF THE ASSEMBLY
Dr. Mather Pfeiffenberger, Alton, Ill.

BOARD OF TRUSTEES
Dr. Edwin S. Hamilton, Kankakee, Ill.
Dr. Mather Pfeiffenberger, Alton, Ill.
Dr. Maurice R. Williamson, Alton, Ill.
Dr. Henry G. Langworthy, Dubuque, Ia.
Dr. John E. O'Keefe, Waterloo, Ia.
Dr. Tom B. Throckmorton, Des Moines, Ia.
Dr. John M. Dodd, Ashland, Wis.
Dr. Eugene S. Sullivan, Madison, Wis.
Dr. Stephen E. Williams, Chippewa Falls, Wis.

EDITOR-IN-CHIEF, POSTGRADUATE MEDICINE
Dr. Charles W. Mayo, Rochester, Minn.

COMMITTEE ON MEDICAL RESEARCH AND ADVANCEMENT
Dr. Elliott P. Joslin, Boston, Mass.
Dr. W. Wayne Babcock, Philadelphia, Pa.
Dr. Charles H. Best, Toronto, Ont.
Dr. George Crile, Jr., Cleveland, Ohio
Dr. Evarts A. Graham, St. Louis, Mo.
Dr. Roy D. McClure, Detroit, Mich.
Dr. Jonathan C. Meakins, Montreal, Quebec
Dr. Eric Oldberg, Chicago, Ill.
Dr. James E. Paullin, Atlanta, Ga.
Dr. Erwin Schmidt, Madison, Wis.
Dr. Waltman Walters, Rochester, Minn.

PROGRAM COMMITTEE
Dr. Russell L. Cecil, New York, N. Y.
Dr. Henry A. Christian, Boston, Mass.
Dr. John R. Fraser, Montreal, Quebec
Dr. Duncan A. L. Graham, Toronto, Ont.
Dr. Herman L. Kretschmer, Chicago, Ill.
Dr. Frank H. Lahey, Boston, Mass.
Dr. Charles F. Martin, Montreal, Quebec
Dr. Charles W. Mayo, Rochester, Minn.
Dr. Howard C. Naffziger, San Francisco, Calif.
Dr. Charles H. Phifer, Chicago, Ill.
Dr. Leonard G. Rowntree, Miami, Fla.

The Medical Bookman

PROGRESS IN CLINICAL ENDOCRINOLOGY*

EXTRAORDINARY discoveries in the field of endocrinology in recent years make especially desirable a volume in which authorities in various aspects of this subject express the progress that has occurred up to the present time. Dr. Samuel Soskin, who has specialized in this field for many years and who is Director of the Medical Research Institute of Michael Reese Hospital in Chicago, has undertaken the editing of a volume with many contributors each of whom considers some single aspect of the functions or disturbances of one of the glands of internal secretion. Doctor Soskin himself opens the volume with a consideration of the role of the hormones in metabolism, after which comes an essay by Dr. Abraham White of California on the "Endocrine Regulation of Protein Metabolism." The contributors are chosen from every section of the United States.

THYROID

The section on "Thyroid" opens with a debate between Dr. J. F. McClendon who is convinced that a deficiency of iodine in waters and in food brings on endemic goiter, and Dr. Isidor Greenwald who is just as convinced that it does not. It would appear that most of Doctor Greenwald's evidence is evidence by exclusion.

Other articles concern the relation of nodular goiter and thyroid carcinoma, the thyrotoxic myopathies—an aspect of thyrotoxicosis that is often overlooked. Then come deficiencies of the thyroid in children, usually leading to cretinism. Dr. Saul Hertz, who deserves great credit for his persistence in the belief that radioactive iodine can control practically all diseases of the thyroid, explains the steps in research which has now been generally accepted. He does, however, suggest that no patient with thyrotoxicosis should be treated with radioactive iodine without a preliminary tracer dose and determinations of up-take and excretion.

Dr. E. B. Astwood, who has done much of the basic research on the use of thiouracil, says that both radioactive iodine and the antithyroid drugs are superior to older forms of treatment. He believes that surgical treatment does not have any advantage over radioiodine, which he concedes also has advantages over antithyroid compounds. If it were possible to recognize the patients with hyperthyroidism who might recover from the disorder with little antithyroid therapy, the antithyroid drugs would be the method of choice. Most surgeons are now willing to concede the limitations on surgery brought about by these new discoveries.

Dr. Elmer C. Bartels points out that surgery is cheaper in the long run, surgery is more certain, and, in his opinion, surgery is safer.

THE PARATHYROID

Our basic knowledge of parathyroidism is hardly more than twenty-five years old. Now we know that excessive action of the parathyroids may lead to bone diseases, stones in the kidney and gastrointestinal manifestations. The treatment of hyperparathyroidism is surgical. On the other hand, the treatment of hypoparathyroidism involves the use of the hormone of the parathyroids coupled with calcium and low phosphorus and coupled also with dihydrotachysterol or large doses of vitamin D.

THE ADRENAL CORTEX

As might be expected, the star of any modern textbook on endocrinology is the adrenal gland. Here research has been so extensive in recent years that the rate of advancement has been phenomenal. The adrenal cortex is considered the "effector" tissue of the pituitary. The bibliography of the adrenal is always tremendous and constantly advancing. The relationship of the adrenal to metabolism, to the action of the sex glands, and to the regulation of water and salt makes it truly a vital organ, and the control of the adrenal by the pituitary makes the pituitary truly the master gland.

One of the most important chapters is that of Dr. Joseph C. Jaudon of the Washington University School of Medicine on "Diagnosis and Treatment of Adrenal Insufficiency in Infants." Unquestionably many infants with symptoms of an-

*Progress in Clinical Endocrinology. Samuel Soskin, M.D., Editor. 641 pages. 1950, Grune & Stratton, New York. $10.00.

orexia, regurgitation, vomiting, and a tendency to unexplained dehydration might have been saved with the present knowledge of the relationship of the adrenal to this syndrome.

The cortex of the adrenal yields a variety of steroid hormones concerned with water and the electrolyte metabolism, the carbohydrate and protein metabolism, and the sex functions. These three groups of activities are independent of one another which would seem to simplify but actually complicates the consideration.

In view of the massive literature now developing in this field, the chapter on clinical tests for adrenal cortical reserve by George W. Thorn and Peter H. Forsham brings the consideration closely into clinical practice. The tests that are offered make it possible to diagnose diseases and deficiencies of the pituitary-adrenal system. The adrenal medulla which used to be the star of the adrenal system now takes second place.

Almost as important in space allotment as the adrenals is the pancreas. Fortunately the discovery of insulin stimulated our progress and our research on the pancreas and many leaders in this field contribute chapters which cover completely our knowledge of diabetes. The contributors to this section include not only some of the names of those who have for a quarter of a century led in this field but also some of the younger contributors who will continue progress on the work in the next generation.

From the section on pancreas, this book proceeds to our new knowledge of the ovary and the testis; the complex physiology of menstruation is analyzed and the clinical recommendations lead to the maintenance of normal endometrium, and those aspects of sex endocrinology which affect lactation, hirsuties, and the liver.

Sections are devoted also to the anterior pituitary and the posterior pituitary, but perhaps because of the timing in the development of this book, relatively little attention is given to ACTH. Dr. Ralph H. Kunstadter reports success in stimulating growth of young boys by the use of testosterone propionate and methyltestosterone. Less favorable growth stimulation occurred in true drawfs. Little work has been done thus far in the use of hormones in promoting height of girls.

A review of Simmonds' disease and anorexia nervosa does not seem to recognize the most recent research involving the use of ACTH for overcoming anorexia nervosa.

A special chapter is a report on the use of ACTH and Cortisone in rheumatoid arthritis by the Rheumatoid Arthritis Research Group at Michael Reese Hospital. This chapter indicates clearly that the authors were not ready at the time of its writing to draw any positive conclusions. The chapter is useful in supplying information as to the dosages of the hormones that may be used.

M. F.

New Books Received

► **ELECTROCARDIOGRAPHY; Fundamentals and Clinical Application - - -**

By Louis Wolff, M.D., Visiting Physician, Consultant in Cardiology and Chief of the Electrocardiographic Laboratory, Beth Israel Hospital; Associate in Medicine, Harvard Medical School. 187 pages, with 110 figures. 1950, W. B. Saunders Company, Philadelphia & London. $4.50.

This small primer contains the best available elementary presentation of the basic principles of electrocardiography. The text devotes its first eighty pages to a lucid presentation of the electrical phenomena responsible for production of the normal and abnormal electrocardiogram. In this section the discussions of muscle contraction, the cell membrane, depolarization, the properties of volume conductors, the electrical field around the heart, repolarization, and the anatomy and physiology of the heart lead very easily and naturally to a consideration of unipolar and bipolar patterns in cardiac health and disease.

Dr. Wolff's superb simplification makes this the textbook of choice for beginning students. His discussion of the unipolar precordial and limb leads precedes the consideration of the bipolar standard limb leads, which is the rational approach. The section on clinical electrocardiography is a brief one hundred pages, yet manages to cover more than adequately the subjects of the normal electrocardiogram, bundle branch block, ventricular hypertrophy, coronary heart disease, myocardial infarction, arborization block, pericarditis, pulmonary embolism and Wolff-Parkinson-White syndrome. The illustrations are excellent.

E. B. C.

► HORMONES IN CLINICAL PRACTICE - - -

By H. E. Nieburgs, M.D., Research Associate, Department of Endocrinology and Assistant Professor, Department of Oncology, University of Georgia; Lately Research Endocrinologist, Department of Obstetrics and Gynecology, British Post-Graduate Medical School, and Clinical Assistant, Endocrine Clinic, Westminster Hospital, London. 388 pages, with 57 figures. 1950, Paul B. Hoeber, Inc., New York. $5.50.

The field of endocrinology is continually being enriched with new data, clinical and experimental, some confirming previous results and hypotheses, others contradicting them. A survey of the vast and ever-growing literature is necessary for the busy practitioner, wherein new data are synthesized, and outmoded concepts discarded. To this end, Dr. Nieburgs submits this volume.

In discussing each hormone, a pattern is set forth to which the material can be suitably fitted. The sequence includes the histology of the gland, the physiology of its secretion, relationships with other hormones or factors, sources, standardization, and clinical preparations, applications in practice, methods of administration and dosage. Although the material is very succinctly presented, a most extensive and up-to-date bibliography follows each chapter and represents a point of departure for further reading. The format lacks a certain serenity, but this is a consequence of striving for brevity.

In addition to chapters dealing with each of the known hormones are sections devoted to environmental influences, psychosomatic endocrinology, and vitamin-endocrine relationships.

An exhaustive chapter on Diagnostic Procedures includes a number of excellent photomicrographs of vaginal and endocervical smears. Probably the most distinctive feature of the entire book is the concluding chapter. This is devoted to a listing in tabular form of all the commercial preparations available in America, Britain, or both, presenting information on potency, units, effects, and duration of activity by all the different routes of administration.

This volume is presented not as yet another textbook of endocrinology but rather as a work of reference embodying the most recent data on the physiology of the hormones and their use in clinical practice. As such it will be a useful guide in a difficult and far from completely charted territory.

N. S.

► DIFFERENTIAL DIAGNOSIS OF CHEST DISEASES - - -

By Jacob Jesse Singer, M.D., F.A.C.P., F.A.C.C.P., Medical Director, Rose Lampert Graff Foundation, Beverly Hills; Consultant in Chest Diseases, Cedars of Lebanon Hospital, Los Angeles. 344 pages, with 171 illustrations. 1949, Lea & Febiger, Philadelphia. $7.50.

This presentation was written primarily as an aid to the general practitioner to serve as an approach to the diagnosis of diseases of the chest. Every major thoracic abnormality is covered systematically and coherently. Each section is concluded with an inclusive differential diagnosis that serves to differentiate the abnormality under discussion from other diseases with which it might be confused.

The book is divided into five main categories. The first deals with Diagnostic Methods routinely employed, including the history, examination, laboratory methods, mechanical aids and x-ray. Subsequently the diseases are discussed under the following four main categories: Thoracic cage, Pleura, Mediastinum, Diaphragm and Tracheobronchial tree and lungs. Each clinical entity includes etiology, pathology, clinical symptoms, physical signs, x-ray findings, laboratory tests, and differential diagnosis. A short bibliography is included at the end of each clinical discussion.

This book is not intended as a text, nor as a complete book of reference. The differential diagnosis is not necessarily complete, but the most important practical points are covered. One might like to find included a differential discussion on esophageal disorders, dysphagia, diverticuli, and congenital defects involving the esophagus and trachea (or bronchi). The functional diseases of the lungs are omitted, i.e., bronchial asthma, asthmatic bronchitis, cor pulmonale and pulmonary hypertension. Of course, cardiac disorders are not meant to be included in the scope of this book, although now and then an occasional reference is made to a cardiovascular defect that may enter into a differential diagnosis.

R. W.

► UROLOGICAL SURGERY - - -

By Austin Ingram Dodson, M.D., F.A.C.S., Professor of Urology, Medical College of Virginia; Urologist to the Hospital Division, Medical College of Virginia; Urologist to Crippled Children's Hospital; Urologist to St. Elizabeth's Hospital; Urologist to St. Luke's Hospital and McGuire Clinic, Richmond, Virginia. With contributions by 12 other authors. Ed. 2. 855 pages, with 645 illustrations. 1950, C. V. Mosby Company, St. Louis. $13.50.

This is a thoroughgoing revision and expansion of Dodson's deservedly popular first edition of the same name. It is beautifully illustrated and covers the various operations of urology pretty completely. In addition there are chapters on Anatomy, Urography, Pre- and Postoperative Care, Radiation Therapy, Acid-Base Balance and Fluid Administration, Blood Transfusion and Shock, and Anesthesia.

This work is very useful for the urologic resident and urologic surgeon, and for the general surgeon who does urologic operations on occasion. There are many references to the literature. The reviewer has had occasion to refer frequently to the first edition, and regards the second as still more valuable, especially because it goes into considerable detail concerning the technic of the operations discussed.

One could wish that proof reading, particularly of proper names, had been a little more searching, and that the reader were offered information concerning more types of ureterosigmoidostomy, as well as a more detailed discussion of transurethral resection. However, it is warmly recommended.

C. D. C.

► MITCHELL-NELSON'S TEXTBOOK OF PEDIATRICS - - -

Edited by Waldo E. Nelson, M.D., Professor of Pediatrics, Temple University School of Medicine; Medical Director, Saint Christopher's Hospital for Children, Philadelphia With the collaboration of 63 contributors. New, 5th edition. 1,658 pages with 426 illustrations, 19 in color. 1950, W. B. Saunders Company, Philadelphia & London. $12.50.

The 1950 edition of this book is the fourth revision over a period of twenty years. A comparison of this volume with the first one written by J. Grozier Griffith of Philadelphia more than twenty years ago reveals the tremendous progress this youngest of all specialties in medicine has made in the past two decades.

In the early days discharging ears in the child were the doctor's nemesis. Resisting all forms of known treatment, more often than not they resulted in deafness and surgical mastoids. Today the paracentesis knife lies dormant in the doctor's bag and mastoidectomy is a rarity. In times past the prognosis in scarlet fever, pneumonia, septicemia, and epidemic meningitis was always in doubt. Today with the use of the several antibiotics, the general practitioner and the pediatrist have become conquering heroes. The physician has lost his dread even of such assassins as tuberculosis, influenza, and streptococcic meningitis.

Formerly the differential diagnosis of congenital heart diseases was between a patency and a stenosis, the treatment was a placebo, and the prognosis was good or bad. Now with the aid of the electrocardiograph, the x-ray, and catheterization, and with special tests which measure the oxygen consumption and saturation of the blood, a clear cut anatomic diagnosis can be made which justifies those surgical procedures that may eradicate the defects or provide sufficient relief to enable the patient to live without misery and suffering.

These are only a few of the hundreds of pediatric advancements to be found in this 1700-page reference book by Dr. Waldo Nelson and his 63 collaborators.

To the medical student who is about to gaze for the first time upon the vista of pediatrics and who wonders why the diagnosis and treatment of diseases of children should be set apart as a field separate from general medicine, the chapters on "The Medical Problems of Infants and Children," and "The Physical, Mental, and Emotional Growth and Development of the Normal Child" should be read and re-read at the very outset. The general practitioner who provides about 75 per cent of the medical care of children and who devotes about one-third of his time to pediatrics, should have this excellent text-book at his elbow for daily use. Finally for the teacher of pediatrics, there are new definitions of terms, new diagnostic procedures, new diseases and new forms of therapy which will modernize and make more effective his teaching.

M. S.

► DISEASES OF THE FOOT - - -

By Emil D. W. Hauser, M.S., M.D., Associate Professor of Bone and Joint Surgery, Northwestern University Medical School. New, 2nd edition. 415 pages with 195 figures. 1950, W. B. Saunders Company, Philadelphia & London. $7.00.

Revised to offer an up-to-date version of disorders of the foot and to include the use of the newer antibiotics as their need demands, this book presents a systematic accumulation of data in a lucid manner. Written primarily for general practitioners and orthopedic surgeons, the monograph includes all phases of foot deformities, diseases, anomalies, postural and circulatory disturbances, fractures, dislocations, sprains, specific infections, tumors, and other less frequently encountered but equally important conditions.

The author stresses the fact that basic concepts of treatment have remained unaltered and that all therapy for foot disorders has the paramount intention of retaining function. Disorders affecting infants, children, adolescents, and adults are discussed from a diagnostic, conservative therapy, and operative standpoint. This portion assists one in distinguishing which diseases and deformities are most likely to respond in a satisfactory manner, and aids the practitioner in referring patients where specialized surgical procedures might offer the best opportunity for relief. Almost two hundred clearly reproduced x-ray prints and photographs help to delineate the problems of foot care, and convey to the reader the importance of accurate diagnosis before therapy is instituted.

In addition to a comprehensive review of the entire field, emphasis is placed on the more recent ideas of treatment and care of such entities as acute thrombophlebitis is modernized with suggestions on the use of anticoagulants and antibiotics.

Significantly this new volume is released when foot disorders have reached an all time peak. The approach to therapy is not too technical to discourage the physician who has minimal interest in foot disorders, but who is confronted with these problems during every day practice. With this bias, therefore, towards the simple effective means of providing relief, the book is highly recommended for the general practitioner, the military and industrial surgeon, and others whose contacts are reflected in this field.

H. O'P.

► QUINIDINE IN DISORDERS OF THE HEART - - -

By Harry Gold, M.D., Professor of Clinical Pharmacology, Cornell University Medical College; Attending-in-Charge of the Cardiovascular Research Unit, Beth Israel Hospital; Attending Cardiologist, Hospital for Joint Diseases, New York. 115 pages. 1950, Paul B. Hoeber, Inc., New York. $2.00.

Any monograph on therapeutics written or edited by Harry Gold is certain to be a "Gold mine" of practical advice derived logically from basic considerations in pharmacology. The present volume is no exception.

In 26 brief chapters the author covers the subject of quinidine, its pharmacology, dosage, and clinical application in the following disorders of cardiac rhythm: Premature contractions or extrasystoles (auricular, nodal, ventricular); paroxysmal auricular tachycardia; nodal tachycardia; auricular flutter; auricular fibrillation; ventricular tachycardia; ventricular fibrillation.

Many gems will be found by the careful reader of this book. Proper emphasis is placed on the necessity for observing patients clinically and electrocardiographically while on quinidine, lest early signs of toxicity be missed. On the other hand the enormous tolerance which some patients have for this drug is pointed out. One such patient with hypertensive and arteriosclerotic heart disease took a daily dose of 4 gm. of the drug for the control of a paroxysmal disorder of rhythm for fourteen years and in this period consumed about 20 kilograms of quinidine.

It is the considered opinion of this reviewer that every general practitioner and cardiologist who uses quinidine for the control of disorders of cardiac rhythm would be well advised to read this book before prescribing for his next arrhythmia patient. The volume is, as advertised, a practical manual for all who treat heart disease.

E. B. C.

► BRAIN AND BEHAVIOUR; Induction as a Fundamental Mechanism of Neuro-Psychic Activity - - -

By N. E. Ischlondsky, M.D., New York. 182 pages, with 46 illustrations. 1949, C. V. Mosby Company, St. Louis. $7.00.

In this monograph the author proposes that a process of "induction" is one of the fundamental mechanisms of neuropsychic activity. Induction is defined as follows:

"Every manifestation of a nervous process leads, under certain well-defined conditions, to the appearance of an opposite process. Thus a focus of excitation gives rise to a focus of inhibition—negative induction, and vice versa, a primary inhibitory process results in the appearance of a secondary excitatory focus—positive induction." A stimulus induces an opposite response.

The author describes in detail a series of investigations supporting the above theories. Visual phenomena, especially the formation of after images, are interpreted as demonstrating induction. The suppression of pain by diverting the subject's attention with other stimuli such as a different pain, physical effort, etc., is another example.

Various phenomena observed in human behavior are offered as further proofs of induction. Some examples are the negative response of children to requests of their parents, the urge to defy authority, the heightened desire for objects when they become scarce, and opposition to new ideas. The author concludes with some general statements regarding the psychologic and sociologic significance of the mechanism of cerebral induction, the objectives of which are to adapt, regulate, and protect the individual and his behavior.

W. S. C.

► FUNDAMENTALS OF OTOLARYNGOLOGY; A Textbook of Ear, Nose and Throat Diseases - - -

By Lawrence R. Boies, M.D., Clinical Professor of Otolaryngology, Director of Division of Otolaryngology, University of Minnesota Medical School, and Associates. 443 pages with 184 figures. 1949, W. B. Saunders Company, Philadelphia and London. $6.50.

This is essentially a guide book for beginners, particularly for medical students in their third year of instruction. It is supplementary to clinical teaching and is, therefore, an aid to the instructor. The commonly accepted, present-day plan of joint authorship is followed here, and while the author has written most of the chapters, his eight associates have written the chapters of especial interest to them.

The antibiotics have done so much to revolutionize the entire subject of ear, nose, and throat that one turns to this chapter at once, but finds only four pages devoted to a discussion of the relative merits of these new agents. "Penicillin and streptomycin are the most effective of the antibiotics." The latter is, of course, relied upon for its effects upon gram-negative organisms which are not damaged by the penicillin group. The sulfas have been overshadowed by the molds, but are valuable as synergists. Streptomycin affects both gram-positive and gram-negative organisms but is quickly resisted by them and may soon become inert. It seems to have an unfortunate selectivity for the auditory nerve, causing temporary deafness in the cochlear branch and more or less permanent ataxia when it involves the vestibular branch.

An interesting feature of this book is the "purpose" of doing thus and so. The student is told *why*, and that is an important word in all teaching, if the answer can be given correctly.

I. W. V.

► UNIPOLAR LEAD ELECTROCARDIOGRAPHY - - -

By Emanuel Goldberger, B.S., M.D., Adjunct Physician, Montefiore Hospital, New York; Cardiographer and Associate Physician, Lincoln Hospital, New York; Consulting Cardiologist, St. Joseph's Hospital, Yonkers, New York; Lecturer in Medicine, Columbia University. Ed. 2. 392 pages with 221 illustrations. 1949, Lea & Febiger, Philadelphia. $7.50.

Since Dr. Goldberger's original work in this field, the initial mixed emotions with which the medical profession greeted its publication have gradually given way to acclaim. As further proof

of this we see widespread adoption of augmented unipolar lead electrocardiography and referral to it as "Goldberger leads."

In any new and great discovery, the early years see the accumulation of much information and, from this, changes result. Recognizing this, the author includes in this second edition a major part of the original text but, furthermore, sets forth much new material since the original publication, both of his own studies and recent reports of other investigators.

Outstanding among the innovations are new criteria for abnormality of the Q-wave, new differential features of pulmonary embolism and posterior infarction, and the differentiation of right and left ventricular strain from right and left ventricular hypertrophy.

An entire new section, Normal and Abnormal Cardiac Rhythms, has been incorporated into the text. One of the most valuable additions is a lucid, comprehensive chart showing the six unipolar leads and the three augmented unipolar extremity leads of normal persons and of all the common abnormalities.

N. S.

► CLINICAL BIOCHEMISTRY - - -

By Abraham Cantarow, M.D, Professor of Biochemistry, Jefferson Medical College; and Max Trumper, Ph.D., Commander, H(S), USNR, Lecturer in Clinical Biochemistry and Basic Science Coordinator, Naval Medical School National Naval Medical Center, Bethesda, Maryland. Ed. 4. 642 pages with 38 figures. 1949, W. B. Saunders Company, Philadelphia & London. $8.00.

The three previous editions of this book have been pre-eminently successful in indicating "the manner in which the physician may best avail himself of information which can be obtained by biochemical studies." Accordingly, it has become one of the most widely used texts in clinical medicine.

The format is unchanged; illustrations are few but the simplicity and clarity of the text require little enlargement. The retention of the material is simplified by the frequent numerical listings, and the excellent "Outline of Chemical Abnormalities in Various Disorders."

The dynamic nature of present-day biochemistry results in frequent additions to, and alterations of, established concepts. The authors have extensively revised most chapters. Outstanding among the newly added material are the sections on the fatty liver, the alarm reaction, and goitrogenic agents. The normal chemical standards listed on the inside of the back cover are compact and helpful; a typographical error is the value for the standard urea clearance which should read 54 cc. instead of 75 cc.

N. S.

Books received will be acknowledged in this department each month. As space permits, books of principal interest to our readers will be reviewed more extensively.

Anxiety in Pregnancy and Childbirth. By Henriette R. Klein, M.D., Associate in Psychiatry, Columbia University College of Physicians and Surgeons, New York, Howard W. Potter, M.D., Professor of Psychiatry, Long Island College of Medicine, Brooklyn, and Ruth B. Dyk, M.S., Research Department, New York City Youth Board. 111 pages. 1950, Paul B. Hoeber, Inc., New York. $2.75.

Handbook on Aerosols. By the United States Atomic Energy Commission. May be obtained from Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C. $0.60.

The Ethical Basis of Medical Practice. By Willard L. Sperry, Dean of the Harvard Divinity School. With a foreword by J. Howard Means, M.D. 185 pages. 1950, Paul B. Hoeber, Inc., New York. $2.50.

The What, When, Where of Hay Fever. By the Botanical Research Department of Abbott Laboratories under the direction of Oren C. Durham, chief botanist. May be obtained free by writing to Botanical Research Dept., Abbott Laboratories.

Aetios of Amida; The Gynaecology and Obstetrics of the VIth Century, A.D. Translated from the Latin edition of Cornarius, 1543, and fully annotated by James V. Ricci, M.D., Clinical Professor of Gynaecology and Obstetrics, New York Medical College; Attending Gynaecologist, City Hospital, New York; Consultant in Gynaecology and Obstetrics, Beekman-Downtown Hospital, New York. 215 pages. 1950, The Blakiston Company, Philadelphia & Toronto. $7.00.

Sex Questions and Answers; A Guide to Happy Marriage. By Fred Brown, Ph.D., Clinical Psychologist, The Mount Sinai Hospital; Adjunct Associate Professor of Psychology, New York University, and Rudolph T. Kempton, Ph.D., Chairman, Department of Zoology, Vassar College. Foreword by Abraham Stone, M.D. 264 pages. 1950, Whittlesey House, McGraw-Hill Book Company, Inc., Philadelphia, London & Toronto. $2.95.

Men of Medicine

Dr. John From the Hills

Dr. John Zahorsky, the venerable St. Louis pediatrician, generally acknowledged to have given some forty years ago the first clear clinical description of roseola infantum, always has taken rare pride in his name.

In at least two old-world tongues, Polish and Czech, "Zahorsky" or a reasonable facsimile thereof means "mountain family" or "family from the hills." As the veteran pediatrician views it, that logically makes him "Dr. John from the hills."

Which is just as Dr. Zahorsky would have it. Just three years ago, he abandoned a still-booming St. Louis practice which he shared with his son and successor, Dr. Theodore Zahorsky, to go back to the Ozark hill country of Missouri where his Bohemian-immigrant parents took him to live when he was a boy of seven.

The death of his beloved wife, Beth, to whom he would have been married fifty years this June, did not deter him. "I'm going into the woods," he announced. He quit his post at the Bethesda General Hospital in St. Louis, which he had headed for twelve years and been associated with for almost fifty, and stepped out as pediatrics chief at the St. Louis University Medical School. With an emeritus fellowship from the American Academy of Pediatrics, and the farewell plaudits of the St. Louis medical fraternity to help him keep warm, he headed for the tiny highland hamlet of Steelville, Missouri, to take up residence in Pleasant Valley, in a stone mansion he'd had built forty years ago for just such an eventuality.

But, as the recent publication of his autobiography, "From The Hills,"* helps to attest, he didn't go to the Ozarks to vegetate. Nor even to be content with a batch of published memoirs.

Three times weekly, he goes into town to help the county's old general practitioner with the latter's child patients. Out of 300 cases, only four have had to be sent off to a bigger town for hospitalization. Already, the 79-year-old pediatrician has met a small epidemic of glandular fever, scoring a perfect patient recovery record, without benefit of blood tests or other laboratory procedures but with the help of a few sulfonamide dosages and that long-cherished principle of his—"the most presumptive diagnosis."

John Zahorsky, in brief, is out to prove what he never could do in almost a half-century of city practice—that much of the current propaganda for more hospitals is "poppycock." That "gadgets" may be necessary to the study of certain obscure diseases but can be dispensed with by the practitioner working in a county of 12,000 souls or so. And that, finally, the pediatrician's place still is, as woman's once used to be, in the home.

What is the solution to crowded offices and prevailing medical economics which keep the average pediatrician from learning the complete home pattern of every child in his patient files?

"Work harder!" cries Dr. Zahorsky. It is not at all unlikely that medical journals may hear again from this little maverick with the homespun streak whose career has been a sterling lesson in love of children, love of work, love of the old-fashioned virtues and the willingness to go to bat for a principle, come hell or the disapproval of a large part of the profession.

In 1904, chiefly on the basis of his valuable experience in a St. Louis foundling home, he published "in a magazine not generally available in medical schools" his conviction that "colds" in children were due not to exposure but to contagion. Inability to spread the notion became his first pronounced frustration. It was destined to be one of many in which he, and those who thought like him, have been nobly acquitted with the passing years.

A second time in 1904, he braved the frowns of some colleagues when he took over the medical supervision of an incubator-baby exhibit at the St. Louis World's Fair. He accepted the post, he has since said, only after assurances of top-notch care and equipment; the experience resulted in a publication, "Baby Incubators," which Dr. Zahorsky still considers his best written work.

Observations on the Fair "Pike" and in the Bethesda Foundling Home convinced him that separation of new-born babes from their mothers, and their assemblage in one large room, no matter how aseptic the conditions, ran contrary to every principle of efficiency, logic and psychology. With a wisdom clearly traceable to his own rural upbringing, he argued that the crying and oral rooting of an infant stimulated the flow of mother's milk, while the practice of keeping the infant elsewhere, and of giving it a night-time bottle to "save" the mother, could lead only to the virtual disappearance of natural feeding.

*C. V. Mosby, St. Louis, 1949.

Dr. John Zahorsky in his library at "Silverwood," his present Ozark retreat.

In 1905, in the St. Louis Medical Review, he went a step further. To permit the disappearance of the cradle and its soporific, gentle swaying motion, to discourage rocking, he warned, would rob the infant of a great psychologic boon.

DR. Zahorsky finds himself and other pediatricians fully vindicated in the matter of rooming-in, and is still battling for vindication in the realm of the cradle. "Some day, I believe," he concluded a *Journal of Pediatrics* paper on the subject in 1934, "it will be no disgrace to rear the young baby in a cradle and even sing him to sleep by a lullaby."

In 1910, his attention was attracted to the growing enthusiasm among nose and throat men and among an increasing number of pediatricians for tonsillectomies.

"Articles were published in the magazines, and the fathers and mothers warned that rheumatism, heart disease, anemia, deafness and mental stupidity were caused by diseased tonsils," he recalls.

In 1919, after a five-year study among his own patients, John Zahorsky stood up before a meeting of the pediatrics section of the A.M.A. to suggest that only a greater susceptibility to pneumonia argued in favor of tonsil removal.

Otherwise, he said, the removal of tonsils did not show any permanent benefit. When some col-

leagues responded to this by trying to talk him down, he recalls in his book, he lost his temper and suggested that some members would rather have a good fee than practice scientific medicine.

"So the meeting broke up in a row. I am sorry!" Dr. Zahorsky says these days. The section refused to print his paper in the proceedings—but two years later, out came Dr. A. D. Kaiser with what Zahorsky calls "his monumental work," in which Kaiser, on the basis of 5,000 cases, reported conclusions almost identical to Dr. John's.

It became another point for the man from the hills. He was able to say in 1933, in the *Journal of the Missouri State Medical Association:* "In studying the recent literature I am glad to find that the tonsil question is becoming in the main a pediatric question in which the age of the child, the family history, the previous history, and the susceptibilities and immunity of the child must be considered."

The old fighter was moved to add, more than a trifle dryly, "For a while, it seemed that this operation would become almost as popular as circumcision in boy babies."

Among a grand total of 150 medical papers, Dr. Zahorsky likes to count also as outstanding his 1943 effort in the New York *Archives of Pediatrics,* reporting on the often-insidious harm done by internal use of petrolatum among children, and his 1915 warning, in the *American Journal of Diseases of Children,* that severe irritation and vesication of the diaper region often resulted from free ammonia in the diapers of children.

"A bacteriologist disputed my conclusions," he says of the ammoniated diaper studies. "So I had no comeback." He tended to his growing practice, wrote more of the "pediatric poetry" with which he has delighted several generations of St. Louis youngsters, and in time, he notes more contentedly than boastfully, he was proved right and the bacteriologist in error.

He may be excused at this late stage in the game for taking a little pride in his roseola infantum studies. They, too, got him into a dignified squabble when he first advanced them before the A.M.A. Section on Diseases of Children in 1913 at a meeting in Minneapolis.

He tells his roseola story in his autobiography. It began when, in 1905, he was called to see a baby with a high fever. "The parents were educated, well-to-do people, so naturally I wanted to make a good impression," recalls Zahorsky; only 10 years before he had borrowed $100 to buy himself a bag and instruments, and he had been on his own for only five years at the time of the visit in question. But unhappily, he had to admit that the cause of the fever was obscure.

When the fever persisted into a third day, Dr. Zahorsky recalls, he called upon Dr. E. W. Saunders, his old teacher at Missouri Medical College (forerunner of the Washington University medical school in St. Louis), of whom it was said, "When a case seemed hopeless, he always had an additional remedy to offer." Dr. Saunders, after a hurried examination, decided the child had a "teething" complaint and cut the gums over two upper central incisors.

But the following day the baby developed a measles-like rash. A day later, the infant was well. Dr. Zahorsky dutifully noted the syndrome, in the next year collected half a dozen similar cases. He published his first paper in 1910 and had 30 cases to back him up when he described "roseola infantum" at the 1913 A.M.A. Meeting.

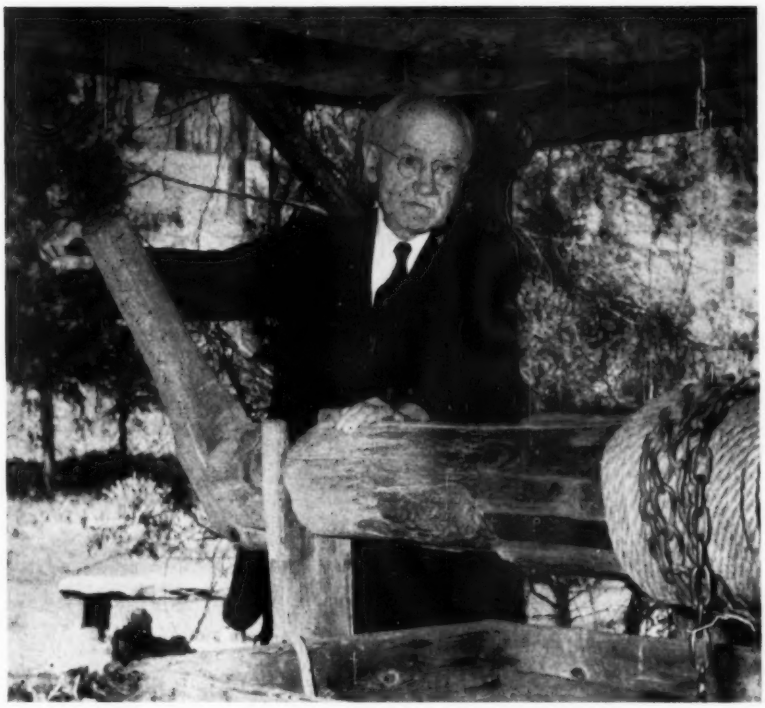
"German measles!" said the scoffers. "Very fanciful. There is no such disease." All but one of the pediatricians in his audience, Zahorsky says, received his report with skepticism. He thought his work was destined for oblivion, until two St. Louis colleagues, Drs. Borden Veeder and T. C. Hempelman, came to his rescue a few months later with a similar report that carried the additional notation of a diminution in white blood corpuscles.

Theirs' and other papers broke the log-jam. "Reports began to accumulate from every state in the union," Dr. Zahorsky writes in pleased reminiscence. "They also emanated from foreign countries. Most of them gave me credit for my discovery: a common disease among infants which, while not fatal, had the most puzzling diagnostic manifestations. Reports from Japan, China and South America followed. It is a world-wide disease among infants."

No doubt, he reflects, the fame which came to him because of this work helped the sale of his lucid "Synopsis of Pediatrics," first published in 1934 and now in its fifth edition. But you can't keep the blunt-talking hill boy in Zahorsky down. "It is one thing to report the discovery of a new disease," he says reviewing the forensic days gone by, "quite another to oppose existing beliefs with another report. From the first event, you make friends; from the latter, some skeptical enemies."

WHAT went into the shaping of this boy from the country who had to work variously as a pharmacist, ladies' wear salesman and file clerk in order to get through medical school—and who tosses off, with the greatest of ease, such neat epigrams as "Pediatrics and economy are inseparable; the proletariat have the most children"?

Dr. Zahorsky begins, himself, with the notation that horoscopically speaking he is a Libran—born (in Mereny, Hungary) on October 13, 1871, which was a Friday. He professes to regard astrology lightly, but he likes to note that Librans are usually reasonable, scientific and strongly attracted to children in their late infancy.



Dr. Zahorsky at the well of the old Ozark homestead where he grew up.

His parents brought him to this country when he was only six months old and they lived for a while in Cleveland before his blacksmith father took his family to the little Ozark farm in Shady Valley. The old log homestead stands there still, over the pike from Dr. Zahorsky's present retreat and surrounded by the green hills in which Zahorsky, a pronounced mystic, finds romance as well as mystery.

John and his brothers attended the one-room log schoolhouse in Pleasant Valley, two up-and-down miles from the homestead. He learned his first and most memorable moral lessons from McGuffey's Reader, and wrote his exercises on a slate with a lead pencil, a tool which remains his favorite to this day. He hunted rabbits and other small game with gun and home-made crossbow, roamed the hills, sat at twilight beside a pond listening to the beeping of the little "peeper frogs." All told, he developed an immense love of home that was to show a direct influence upon his thinking as a physician.

"So many of our people in the cities do not have the chance to develop a love of home, and in this respect their emotional life remains stunted," he was to write, many years later. In 1922, he lectured his confreres in the Missouri State Medical Association: "If the home is the best isolating place against disease, it also is the natural place for the sick child to be treated. When the young child is sick, it is happiest and shows the strongest resistance when under parental care and in its home. The practice of tearing the sick child from its mother's arms and taking it to a hospital, under strange surroundings, looked at by strange faces, administered to by strange methods, and deprived of loving caresses by the parent is not good pediatrics, whether the disease is influenza or poliomyelitis.

"I declare, therefore, that successful pediatrics must maintain and develop the principles and practice of home prophylaxis and therapeutics and not be carried away by the costly idealism of children's hospitals, asylums and clinics. These are only side units to the main show. . . ."

By the same token, Dr. Zahorsky urges today that the undue trend toward specialization be checked, the shortage of skilled doctors in rural areas be cushioned and the curse of what he calls "hospitalism" be scrubbed off young doctors by requiring them to spend five years in general country practice before qualifying for any specialty.

Dr. Zahorsky sees no paradox in the fact that he never practiced medicine anywhere but in St. Louis until his return to the Ozarks in 1947. When he was graduated from Missouri Medical College

in 1895, he says, there was no need for additional physicians in his home area; besides, city practice was more a challenge than a lure. In the first two months of the frustrating period in 1895 when Zahorsky and his father-in-law-to-be, the late Dr. W. F. Silverwood, put out their G.P. shingles at an office in downtown St. Louis, Dr. Silverwood had three patients, only one of whom paid his bill. And Dr. Zahorsky had a total of two patients—income $1.50.

Hero worship with a young Steelville, Missouri, physician, Dr. Charles Arthur, as its object made the medical field an obsessive ambition for him by the time he was 17 and ready to leave home for a clerk's job in a general store at nearby Hawkin's Bank. His father opposed the idea of aspiring to be a doctor.

"Father was afraid I'd lose him his entire acreage in the doing," Dr. Zahorsky muses. "That was in the days when medical school tuition was $60 yearly, the total annual expenditure not more than $300, and a man could get his M.D. for about $1,000 all told."

Zahorsky turned the trick with $500 borrowed from his father and $500 earned in his interim job in the rural general store. "Today, it would cost father his farm and then some," Dr. Zahorsky says, not a little sadly. "Think of it—between $6,000 and $10,000. No wonder a farm boy today can hardly dare let himself toy with the possibility of a career in medicine."

He defends the St. Louis of the '90s against those easterners who, noting the city's 11 medical schools, pronounced it the "Medical Center of Cults." True enough, he acknowledges, all shades of medical quackery were represented. But on the other hand, the expanding West desperately needed doctors, and what many a poorly-trained physician did not get from his schooling, he picked up by actual experience and from reading.

Not so John Zahorsky, of course. Missouri Medical was the mid-west's best entry of the period, and Zahorsky and his mates worked ten hours a day, seven months a year for three years.

It was during his general store career that Dr. Zahorsky witnessed his first operation, when an out-of-town surgeon amputated the crushed fingers of an injured miner, using the store's loading porch as an operating table. Coincidentally, his own first operation also was a finger amputation, performed one day in medical school when he was called upon by one of his more perceptive teachers.

Dr. Zahorsky did well on that occasion and got a nice round of applause, but he didn't like it. "My awkwardness in manual manipulations was well known to me," he says.

During his medical school days, he pretty-well shunned such stage diversions as St. Louis had to offer, but not only because of his pocketbook malaise. "The voices and songs and pretty women deranged my scientific thoughts for several days," he says. "Strong emotional disturbances are to be shunned when one is training as a scientist."

It was ten years before Zahorsky, more or less guided to it by his work at the Bethesda Foundling Home and his period with pediatrician Saunders, upped his home-visit fee from $2 to $3 and changed from practitioner to specialist.

He recalls that decade of O.B. vigils in kitchen chairs, and of immersion in family joys and problems with considerable warmth, that somehow can't be dismissed as an old man's nostalgia. "Alas, the status of the general practitioner has sunk to a low level, and the feeble efforts of medical schools have not succeeded in furnishing an acceptable substitute for him." Dr. Zahorsky feels his years as a practitioner alerted him to the practitioner's greatest strength: the faculty of being suspicious, of refusing to dismiss every disease as simple and innocuous.

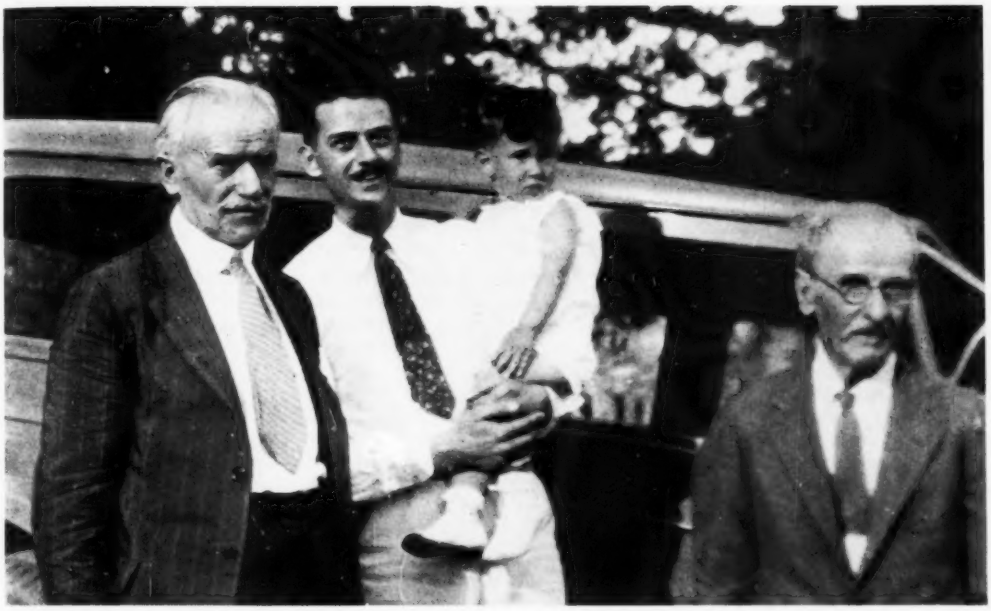
As a pediatrician, Dr. Zahorsky held not only daytime but evening office hours, a policy he was to pursue for more than forty years. He confesses—and no doctor has soul so dead he will not perceive the background for it—that this encroachment on family life led to the only major disagreement he ever had with Mrs. Zahorsky. In those days he sported a beard, but he says this was not an additionally disturbing factor; he soon shaved it off in favor of a fine bushy mustache.

They had a daughter, Elizabeth, now the wife of a college-trained Ozark farmer, and one son, Theodore. Dr. Zahorsky confesses that the boy showed signs in his teens of a bright interest in agriculture and knew, from observing his father, that medicine the Zahorsky way meant few outside pleasures and a defective family relationship. The family's friends "pushed him into it" nevertheless, giving St. Louis another Zahorsky to perpetuate the clan's pediatric tradition.

In the realm of pediatrics, Dr. John Zahorsky stands out for the rare fashion in which he kept pace with new developments even while doggedly holding to such of the old precepts as he felt were worth saving.

Thus, he successfully resisted the lure or overindulgence which came in on the wave of behavior psychology and "personality development." In 1940, he wrote in the *Texas State Journal of Medicine:* "Overindulgence is a species of neglect. Physical, mental and psychic conflicts are inevitable in the process of development. In fact, it is clear that the child who has not been subjected to some stress . . . during childhood can scarcely develop into a normal man or woman."

The best way, in fact, to summarize the work of John Zahorsky is to say that he has resisted fads.



Four generations of Zahorskys (1933) at the Shady Valley homestead near Steelville, Missouri. *From left:* Dr. John Zahorsky, son Dr. Theodore with one of Dr. John's grandchildren, and Dr. John's father, the late John Zahorsky.

"The man of science should beware of popular acclamation, for fashion is the ruling power among the laity. Pediatrics at times suffers severely from popular fallacies. It is fashionable now to have the boy baby circumcised. It is fashionable to wean the baby promptly after birth and feed him evaporated milk from a bottle. It was fashionable to stop rocking the baby and pay no attention to him. It was fashionable to avoid drafts and exposure to a current of air.

"Happily, these fashionable habits are slowly discarded by those pediatricians who know that the welfare of the baby is above pseudoscience and personal prejudices."

Unashamedly, Dr. Zahorsky says the old virtues—mother love and a sane, middle-of-the-road home—are the sincere pediatrician's best trumpcards, even in this stage of an era which he has seen from the beginning, a "speeding cascade of new ideas, new methods and complicated hospital procedures."

In his personal life, Zahorsky never has let his yen for the woods swamp his practical sense. He figures that in forty years his Ozark farmland, operated by renters, has averaged a 3 per cent return on the investment. "That's as much as insurance companies or government bonds pay," he says smartly, "and I would rather own the farms."

These days, he still takes long walks over his beloved hills. Otherwise, he spends time studying and sketching the "little trees"—the myriad varieties of Ozark weeds whose possible medicinal elements intrigue him. Now and then, he may even dash off another bit of pediatric poetry.

Hundreds of Missouri's "Zahorsky children" and many a student of his during thirty-six years at St. Louis University remember some of those jingles, which "Dr. John From The Hills" took the trouble to compose for use in those troubled moments when a child needed a bit more than pharmaceutics to ease its pain.

Humpty, dumpty, dum,
List to my noisy drum.
The titmouse caught a sparrow hawk,
The spider made the lizard talk.
The nighthawk wooed a whippoorwill
Beneath the hawthorn on the hill.
The owl was prowling through the day
To catch a tortoise in the hay.
A mocking bird sat on a box
And shyly flirted with a fox.
A thirsty duckling in the grass
Was making tea from sassafrass.
Humpity, bumpity, bum.

Old admirers of Dr. Zahorsky think it's the rare young pediatrician who has poetry for his patient—or even enough time. They may be right when they say that it takes a man from the hills.

ROBERT SCHULMAN

Consultation Service

This special consultation information service is offered as a regular monthly feature of *Postgraduate Medicine*. Subscribers are invited to call on this Service for answers to difficult medical problems from members of our Editorial Board and our Editorial Consultants best qualified to help. Each question will be answered by mail and those of general interest will be published each month. Address all communications to Consultation Service, *Postgraduate Medicine*, 516 Essex Building, Minneapolis 3, Minnesota.

HEPARIN OR DICUMAROL IN ANGINA PECTORIS

QUESTION: Is it advisable to use heparin or dicumarol in the case of a 65-year-old man with angina as a preventive of thrombosis? Are there any dangers from the use of either?

M.D.—Montana

ANSWER: It is difficult to answer this question without more details than are recited. However, from my own experience and observation I do not think it advisable to use heparin or dicumarol in any patient with angina pectoris with the idea or the hope of preventing coronary thrombosis. There are some dangers connected with the use of both of these remedies provided the prothrombin time of the patient is not accurately followed. And if the patient has hypertension he might have a cerebral hemorrhage, which in the presence of dicumarol or heparin could be made worse.

JAMES E. PAULLIN

CAUSE AND TREATMENT OF VERTIGO

QUESTION: I would appreciate your suggestion regarding the diagnosis of a 52-year-old male, whose family and personal history are unimportant and whose complete workup including physical, special examination of the eyes, ears (including caloric and audiometer, etc.), EKG, skull x-rays, GI and GB series, Wassermann, blood chemistry, are all negative. He is 40 pounds overweight and always has been.

About two years ago he was awakened by a feeling of vertigo which lasted a few moments causing an almost complete blackout accompanied by profuse sweating. Any attempts to move his head or assume the upright position would bring the attack back in a lesser degree. The following day there would be minor episodes of giddiness, a feeling of uncertainty in walking or getting up from the chair or moving the head quickly or even putting on rubbers. Finally in a day or two the situation would clear up and the attack would reappear in a few weeks or months.

It appears that the attacks only come on in the early morning; there is cold, clammy skin with profuse sweating; the vertigo usually is counterclockwise; emotional upsets the preceding day seem to predispose attacks; by holding himself rigid (until the attack builds itself up to a climax and then "pops" as he expresses it), he can shorten the attack. There have been no convulsive seizures nor complete loss of consciousness, and between attacks he feels well, although he has had to resort to fairly heavy doses of sedatives to keep these attacks from recurring often. He also states that large doses of testosterone (from 50 to 100 mg. weekly) seemed to help him. He also stated that he thought that the various phases of his eye examination might have precipitated an attack as he felt "funny" upon leaving the oculist. He does have a bad visual error, since he cannot distinguish one person from another without his glasses at any distance.

M.D.—New York

ANSWER: The condition of the patient whose history and findings are given here may be classified as Ménière's disease without labyrinthine hydrops or serous labyrinthitis. Some authorities, such as Lindsay, however, prefer to classify vertigo without deafness as "pseudo-Ménière's disease."

Lelli has demonstrated that the vestibular nuclei receive part of their blood supply from the internal auditory artery. Therefore, this type of disorder, the result of vasospasm, may be due to anoxia of the labyrinthine nuclei rather than the end-organ.

This syndrome is one of those classed as "focal" or "localized autonomic dysfunctions," and is related to the histaminic cephalgias, myalgias, vasomotor coryza group.

The fundamental pathologic process is vasospasm, but the vasospasm usually involves the arteriole just before it enters the capillary loop. There is disturbance of the normal co-ordination between arteriole and capillary which, according to Krogh, have a separate autonomic nerve supply. As a result, the capillary loop becomes atonic, sludging of the blood occurs and anoxic conditions are produced in the tissues supplied by this loop.

Appearance of symptoms in the early morning hours is frequent because it is at this time that the peripheral vasodilatation during sleep begins to change to the normal vascular tone of the waking hours, and this favors development of autonomic dystonia.

Since the cause of the disorder is vasospasm, the indicated treatment is vasodilatation. Mono-ethanolamine nicotinate given subcutaneously probably is the most convenient vasodilator for office use. This can be administered, 25 mg. the first day, 50 mg. the second and so on, until 100 mg. is being given daily. As a rule this is an effective dose for most patients. Dilatation of the intracranial vessels is approximately parallel to dilatation of the vessels of the facial skin, so the effectiveness of the dose can be judged by the facial flush secured. Some patients are unusually sensitive to vasodilators, and require a smaller initial dose and smaller increments.

Roniacol, a long-acting vasodilator, may be administered by mouth, starting with a half to a fourth of a 50 mg. tablet given three times daily and increasing the dose gradually until 50 mg. is given three times daily. Treatment should be continued for approximately a month.

Since there is an inherited physiologic dysfunction, repetition of attacks may be expected when the patient is again exposed to a preferred stimulus, and treatment must be repeated. Physical agents such as cold, heat, fatigue, emotional perturbations and the like are likely to be the precipitating factors.

ANTIHISTAMINIC DRUGS DURING PREGNANCY

QUESTION: Is there any reason why antihistaminic drugs could not be given during pregnancy?
M.D.—Connecticut

ANSWER: The use of antihistaminic drugs is not contraindicated in the presence of pregnancy. These drugs can cause fatigue and marked lethargy. Early pregnancy may do the same. Hence minimal doses are indicated in early pregnancy.

JOSEPH L. BAER

TREATMENT OF URETHRITIS

QUESTION: I have a patient, an adult male, 27, single, occupation underground iron miner, whom I have been treating for a case of urethritis for almost two years. When he first came in he had the typical yellow purulent discharge of acute gonorrhea. His two-glass test was positive at that time. Since then both glasses have been clear after the first two months of treatment. He complains of a discharge in the morning. At times this disappears entirely, then it recurs. It may be purulent at certain times and at other times it is watery. Most of the time the discharge is very scant; at other times, it is more abundant. I might say that prostatic smears have always been found to be negative for gonococci.

He was treated at first with sulfathiazole. He has also received a great number of injections of penicillin. He has had injection of dihydrostreptomycin and of late has taken aureomycin internally as well as pyridium, also Lafayette's mixture. He has had injections of neo-silvol, also of weak solutions of zinc sulfate. At present, I am trying injections of penicillin given twice a week. At the same time I am giving prostatic massage on the same day. Have you anything further to suggest?
M.D.—Michigan

ANSWER: Evidence, as given in the question, indicates that this patient did have a gonorrheal urethritis, although no cultures were made and there is no description of the Gram stain which should have been made to identify the type of organism present.

I would now obtain a culture of the discharge appearing at the meatus of the urethra. I would also get a specimen of urine after the prostate is massaged, and make a culture from this urine sediment.

After the result of the culture is known, I would investigate this man's sexual partner, and see what type of infection she may have.

It is possible that this patient has been overtreated, and a rest for a month or two might be useful. If active treatment is to be continued I would gradually dilate the urethra, especially the prostatic portion, with sounds, and gentle massage could be made over the prostate gland and the urethra when these sounds are in place. Following the use of the sounds the urethra should have instilled into it some disinfecting solution, or before the sounding is done the bladder should be filled with mildly disinfecting solution, so that following the treatment of the urethra with sounds and massage, the patient may urinate a sufficient amount so that a thorough mechanical cleansing and disinfection of the urethral mucosa may occur.

If the organisms that are causing the urethritis are of a coccus group, they certainly are not susceptible to the ordinary antibiotics that are most effective in treating conditions of this sort. It may be necessary after you know the type of organisms that are present, to test their susceptibility to various dilutions of penicillin, streptomycin, etc.

Every urologist sees cases of "abacterial urethritis" in which a discharge is present but no organisms can be obtained by smear or culture. Sometimes we feel that this may be caused by a virus. When this condition is present, we have used arsenicals to very good effect. This may be used intravenously in moderate doses every four days for three or four doses.

Another condition one must remember is an upper urinary tract infection that may constantly re-infect the prostate gland and the urethra. There is no contraindication to the use of the cystoscope so that a complete genitourinary survey may be made. When a condition of this character does not clear up under adequate treatment you must assume there may be a focus in the kidneys and that catheterization and pyelo-ureterograms must be made, and cultures of the specimens of urine obtained from each kidney.

GILBERT J. THOMAS

TREATMENT FOR LEUKODERMA

QUESTION: A married Chinese lady of 56 years has had a very slowly growing leukodermal patch over her left cheek for fifteen to twenty years. The extension of the white patch is so slow that one can hardly notice any change within a year. The patch is extending across the forehead and also over the left side of the neck. She has received various conventional therapy, except the copper injections, without any effect. Will you please give the modern treatment of this kind of skin disease?
M.D.—New Jersey

ANSWER: At the present writing there is no known curative therapy for leukoderma. Proper dilutions of walnut juice or potassium permanganate solution may be applied locally to temporarily stain the leukodermic areas the color of the surrounding normal skin.

BEDFORD SHELMIRE

New Drugs *and* Instruments

Information published in this department has been supplied by the manufacturers of the products described.

SUCARYL

PURPOSE: For use in diabetic, reducing or other diets in which sugar is forbidden or amount limited.

COMPOSITION: Sucaryl Sodium is the Abbott trade name for sodium cyclo-hexylsulfamate.

DESCRIPTION: Noncaloric sweetening agent in tablet form with the natural sweet taste of sugar. Retains its sweetness in cooking, baking, and canning and boiling solutions, and has no bitter after-taste when used in moderate amounts. One effervescent tablet is equivalent in sweetening power to one teaspoonful of sugar.

DOSAGE AND ADMINISTRATION: It is recommended that adult patients limit their daily intake to 8 tablets (1 gm.). Patients suffering from severe kidney impairment should use on moderate amounts and under medical supervision. Tablets may be added directly to foods being cooked and to hot or iced drinks, or may be crushed and sprinkled on cereals or fruits.

HOW SUPPLIED: In ⅛ gm. grooved tablets, bottles of 100 and 1,000.

PRODUCER: Abbott Laboratories, North Chicago, Ill.

FURASPOR

PURPOSE: Topical treatment of tinea capitis.

COMPOSITION: Eaton brand of nitrofurfuryl methyl ether.

HOW SUPPLIED: Packaged as a 1 per cent concentration in a water-dispersible, nonstaining, odorless vanishing cream base, in 3 ounce jars.

PRODUCER: Eaton Laboratories, Inc., Norwich, N. Y.

FEOJECTIN

PURPOSE: Stable saccharated oxide of iron for intravenous injection.

COMPOSITION: Each 5 cc. ampule contains the equivalent of 100 mg. of elemental iron.

INDICATIONS FOR USE: Cases of iron-deficiency anemia in which oral medication is relatively ineffective, not well tolerated, or produces results too slowly. Particularly indicated for: resistant hypochromic anemias; anemia in pregnancy; anemia in gastrointestinal diseases, bleeding hemorrhoids, or nutritional deficiencies.

DOSAGE AND ADMINISTRATION: Complete dosage data provided in package circular.

CAUTION: As with any intravenous medication, caution should be exercised with cardiovascular patients.

HOW SUPPLIED: Boxes of 6 5-cc. ampules.

PRODUCER: Smith, Kline & French Laboratories, Philadelphia, Pa.

SURGICAL FILM LIBRARY CATALOG

The Surgical Film Library, sponsored by DAVIS & GECK, INC., has released the 19th edition of their catalog. The library contains approximately 150 subjects dealing with various surgical technics performed by the country's foremost surgeons. The films are loaned without charge to hospitals, nurses training schools, medical schools giving advanced courses in surgery, and to other accredited medical and surgical groups.

A branch library is also maintained in Canada with adequate copies of films for distribution through central headquarters in Toronto. A catalog listing the subjects in Canada is also available.

Requests for catalogs, both American and Canadian, should be addressed to Surgical Film Library, Davis & Geck, Inc., 57 Willoughby St., Brooklyn 1, New York.

LIQUID TAKA-COMBEX

PURPOSE: Treatment of patients having faulty B complex intake, particularly when associated with digestive disturbances.

COMPOSITION: Each 4 cc. (1 teaspoonful) contains:

Taka-Diastase (starch-liquefying enzyme)	2½ gr.
Vitamin B_1	2 mg.
Vitamin B_2	1 mg.
Vitamin B_6	0.5 mg.
Pantothenic acid (as the sodium salt)	2 mg.
Nicotinamide	5 mg.

INDICATIONS FOR USE: In convalescence, restricted diets, anorexia, geriatric and pediatric nutrition, during pregnancy and lactation.

HOW SUPPLIED: In 16-ounce bottles.

PRODUCER: Parke, Davis & Company, Detroit, Mich.

LUMINAL SODIUM AMPINS

PURPOSE: Sedative, hypnotic, anticonvulsant.

DESCRIPTION: Each ampin contains 0.324 gm. (gr. 5) Luminal sodium in 2 cc. propylene glycol solution for intramuscular injection. Syringe, ampule, solution and needle all in a single, sterile injection unit ready for immediate use. Remove needle-cover, insert needle and break tip of ampule. Controlled inert gas pressure immediately completes injection.

INDICATIONS FOR USE: Employed principally as a sedative in conditions of excitement of the nervous system; for its hypnotic and antispasmodic action in cases in which oral administration is impossible or impracticable. For use in status epilepticus, vomiting of pregnancy, eclampsia, psychoses, drug addiction; and postoperative medication.

DOSAGE AND ADMINISTRATION: *Intramuscularly* only. Average individual dose for adults ranges from 0.1 to 0.32 gm. (gr. 1½ to 5). Larger doses may occasionally be necessary in status epilepticus, psychoses, pronounced excitement and insomnia in mental cases. However, the total dose should not exceed 0.6 gm. (gr. 10) in twenty-four hours. The effect of large doses must be closely watched, and alkalis should be given and the bowels regulated during prolonged use.

HOW SUPPLIED: Luminal Sodium Ampins, 2 cc., boxes of 5.

PRODUCER: Winthrop-Stearns, Inc., New York 13, N. Y.

STERILJAR

PURPOSE: Double germicidal protection of Scanlan sutures and extreme visibility.

DESCRIPTION: The sterile tubes containing the sutures, deeply immersed in alcohol, are immediately ready for surgery without sterilizing. The Steriljar's plastic screw cap prevents evaporation or leakage in any position. The all-glass jar is reusable as a transparent utility container.

PRODUCER: Ohio Chemical & Surgical Equipment Co., Madison, Wis.

NEW SUTURE-NEEDLE COMBINATIONS

DESCRIPTION: Most of the new items are combinations of Champion-Pare serum-proof silk suture and Gudebrod's Mintraumatic swaged-on needles. Of interest is the introduction of a special combination for intrathoracic blood vessel anastomosis, as well as combinations for nerve, arterial and intestinal surgery.

PRODUCER: Gudebrod Bros. Silk Co., New York, N. Y.

STOLIC FORTE TABLETS

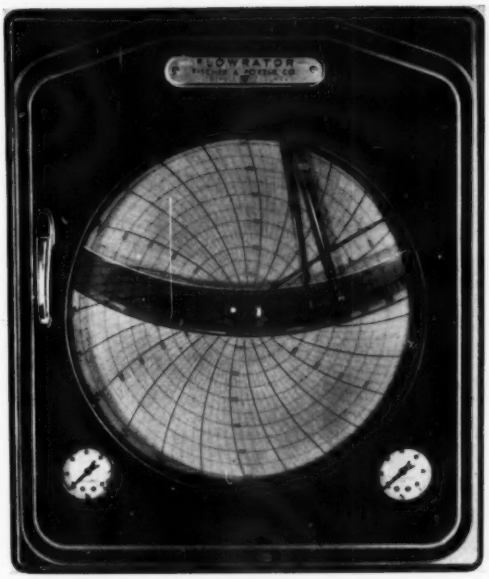
PURPOSE: To provide prolonged vasodilation and effective sedation in the control of essential hypertension.

COMPOSITION: Contain 30 mg. mannitol hexanitrate; 20 mg. rutin; 30 mg. Delvinal vinbarbital.

DOSAGE AND ADMINISTRATION: Recommended adult dosage is from one to two tablets at intervals of from four to six hours.

HOW SUPPLIED: Bottles of 100 and 1,000.

PRODUCER: Sharp & Dohme, Philadelphia, Pa.



RATOTHERM*

PURPOSE: For measurement and control of temperature.

DESCRIPTION: Ratotherm temperature instruments have thermal systems filled with liquid, vapor-liquid, or gas; or combination temperature-sensing and pneumatic-transmitting elements. Standard ranges covered lie between −125° F. and +1,000° F. Temperature readings can be transmitted pneumatically direct from the point of measurement to locations distant 1,000 feet or more.

Several types of capillary tubing are available in many materials of construction, and ambient temperature compensation is provided when necessary. Other measuring units, sometimes up to a total of four, may be added as required—in addition to temperature alone, pressure, flow, speed and motion recorders may be incorporated into the same case.

PRODUCER: Fischer & Porter Company, Hatsboro, Pa.

*Trademark.

DODEX DROPS WITH FERROUS GLUCONATE

PURPOSE: Special blood builder for children.

COMPOSITION: Two 15-cc. vials, one containing vitamin B_{12} (10 micrograms per cubic centimeter), the other containing ferrous gluconate (218 mg. per cubic centimeter—the equivalent of 25 mg. of iron).

DOSAGE AND ADMINISTRATION: Recommended dosage varies with age of infant or child, ranging from 3 drops from each vial once or twice a day for infants aged 3 months, to 10 to 20 drops from each vial once or twice a day for children 2 years or older.

HOW SUPPLIED: Combination package containing two 15-cc. vials, one of B_{12} and one of ferrous gluconate, each with an individual dropper.

PRODUCER: Organon, Inc., Orange, N. J.

No. 40 METAL OPHTHALMOSCOPE

DESCRIPTION: New instrument has one-finger selection of dioptric lenses, aperture, and light intensity, enabling the operator to complete the examination without change of examining position. It has three apertures—standard, pin-hole, and vertical slit; also a red-free filter for neutralizing the blood-red reflex, accentuating minute hemorrhages and va ular twigs, exaggerating fiber bundles and assisti g in determining whether lesions in hemianopsia cases are below or above primary centers.

The dioptric disk has a range of −20 to +25 with a window to protect legibility of dioptric numerals. A double condensing lens with fixed focus provides maximum bright illumination. The housing is metal, thin, light, and dust-proofed, and finished in matt black.

PRODUCER: National Electric Instrument Co., Inc., Elmhurst, L. I., N. Y.

LIRON®-FORTE

PURPOSE: Encourage increase of hemoglobin and red blood cells.

COMPOSITION: Each Caplet contains:

Ferrous sulfate, exsiccated	200	mg.
Liver concentrate, N. F. IX.	240	mg.
augmented with		
Thiamine hydrochloride	2	mg.
Riboflavin	2	mg.
Nicotinamide	10	mg.
Folic acid	1.7	mg.

INDICATIONS FOR USE: When blood-building essentials are required in therapeutic amounts, not merely to reinforce the diet.

DOSAGE AND ADMINISTRATION: One or more Caplets three times a day, after meals.

HOW SUPPLIED: Bottles of 100, 500, and 1,000 Caplets.

PRODUCER: George A. Breon & Co., Kansas City, Mo.

ENTOZYME

PURPOSE: Specially constructed tablet which releases digestive enzymes at different levels in the alimentary canal.

COMPOSITION: Each tablet contains:

Pepsin, N.F.	250 mg.
Pancreatin, U.S.P.	300 mg.
Bile salts	150 mg.

DESCRIPTION: Tablets have an outer shell which, being gastrically soluble, releases pepsin in the stomach to act on proteins; inner core of pancreatin and bile salts are liberated only when core reaches the small intestine, and digests fats, starches, and completes the digestion of proteins.

PRODUCER: A. H. Robins Company, Inc., Richmond, Virginia.

CHOBELINE

PURPOSE: Prevention and treatment of fatty infiltration and degeneration of the liver.

COMPOSITION: Each 30 cc. (approximately 1 fluid ounce) contains:

Choline chloride	2.0	gm.
Thiamine hydrochloride	16.7	mg.
Riboflavin	6.67	mg.
Niacinamide	33.3	mg.
Sodium benzoate as preservative	0.1	per cent

DOSAGE AND ADMINISTRATION: One or two tablespoonfuls with meals three times daily or as directed by physician. Dilute with water if desired.

HOW SUPPLIED: Pint and gallon bottles.

PRODUCER: Reed & Carnrick, Jersey City, N. J.

LUGOLCAPS

PURPOSE: Medical management of hyperthyroidism.

COMPOSITION: Each capsule contains 40 mg. of iodine, part of which is present as free and part as combined iodine. Diglycocoll hydriodide and iodine are used in the capsule in the ratio of two molecules of diglycocoll hydriodide to one of iodine, the formula being $2\ (CH_2NH_2COOH_2).HI+I_2$.

INDICATIONS FOR USE: In producing thyroid involution so that at operation (thyroidectomy) bleeding as a result of vascularity is not encountered. Also used postoperatively in recurrent hyperthyroidism and in mild hyperthyroidism in which medical management is the treatment of choice.

PRODUCER: The Burnham Soluble Iodine Co., Auburndale, Mass.

NORMOCYTIN*

PURPOSE: Treatment of pernicious anemia and other megaloblastic anemias.

COMPOSITION: Each cubic centimeter contains 30 micrograms of a concentrate of vitamins B_{12b} and B_{12} prepared from the fermentation of *Streptomyces aureofaciens*.

HOW SUPPLIED: In packages of 1, 5, or 25 ampules of 1 cc. each.

PRODUCER: Lederle Laboratories Division, American Cyanamid Co., Pearl River, N. Y.

*Trademark.